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VOLUME I NUMBER 1

THE
SURGICAL CLINICS
OF
JOHN B. MURPHY, M.D.
AT
MERCY HOSPITAL, CHICAGO

FEBRUARY 1, 1912

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PUBLISHERS' ANNOUNCEMENT

The Surgical Clinics of Dr. John B. Murphy, at Mercy Hospital, Chicago, of which this is the first number, are to be published bi-monthly, *i. e.*, six times a year. They are the clinics that are held at the hospital every Wednesday and Saturday, for physicians only.

There is, as is well known, a constantly growing demand for strictly clinical teaching, and it is universally conceded that in this Dr. Murphy stands without a peer. The profession is to be congratulated on the fact that Dr. Murphy has consented to allow us to report his clinics and publish them to the profession.

From the profession all over the United States and Europe there has been a great demand to obtain for permanent record the teachings of Dr. Murphy, and it is in response to this demand that the Doctor finally yielded to our desire to place them before the profession.

The history of each case is given, and then follow the comments of Dr. Murphy covering the technic of the operation, the diagnosis, the methods of arriving at the same, and all the points involved in the *living pathology* of the particular type of case.

The publishers desire to emphasize the fact that these are verbatim stenographic reports of the famous clinical talks of Dr. Murphy, and are in no sense to be confused with a systematic book on surgery.

They are edited only for the purpose of eliminating unnecessary repetition; repetitions, however, will be given where they accentuate certain conditions and points in the particular case. The Clinics will be illustrated with skiagrams, photographs, and sketches, wherever necessary.

PUBLISHERS' ANNOUNCEMENT

AMONG THE OPERATIONS AND CLINICAL TALKS THAT WILL APPEAR IN THE APRIL NUMBER OF THE CLINICS ARE THE FOLLOWING:

UNUNITED FRACTURE OF THE TIBIA (Implantation of New Bone)
ARTHRITIS OF KNEE JOINT
UNUNITED FRACTURE OF NECK OF FEMUR
ANKYLOSIS OF KNEE (Arthroplasty)
UNUNITED FRACTURE OF HUMERUS (Transplantation of Bone)
ANKYLOSIS OF HIP (Arthroplasty)
POTT'S FRACTURE
CHARCOT ANKLE

All these cases will be well illustrated with photos and *x*-ray pictures.

SURGICAL CLINICS OF JOHN B. MURPHY

CARCINOMA OF THE BREAST

HISTORY

Female, a Dominican sister, age forty-six; admitted December 4, 1911, because of a growth in the right breast. Family history negative. Past history, diphtheria at the age of thirty; inflammatory rheumatism in 1893; had palpitation and weakness of the heart following the rheumatism. Uses tea and coffee moderately. Menstruation normal.

Present Trouble.—On the twenty-fifth of August, 1911, the patient states that while washing herself in the morning she noticed a small nodule embedded about one inch in the tissue immediately to the right of the nipple. The nodule at that time was hard, and of the consistence and the size of a cherry. It was freely movable, and was not in the least tender on pressure. The patient states that she forgot about the breast, and she did not notice it until the twentieth of November, when she had a burning sensation at the site of the nodule. She examined the nodule and found that since August 25th the nodule had grown to the size of a small egg; this period of three months was marked by no subjective symptoms of any kind. The tumor mass, besides burning her on November 20th, also was the seat of sharp pains, though these were not especially severe. During the time elapsing between November 20th and December 4, 1911, the patient states that the mass has been somewhat tender to pressure, and the burning sensation has been present at intervals. She has felt well in every respect and has lost no weight. On examination a nodule the size of a hen's egg is palpable just to the external margin of the right nipple, embedded in the breast tissues about ¾ inch.

The mass is not tender on palpation; the skin is slightly movable over the mass; there is no retraction of the nipple, no dimpling of the skin, and no discharge from the nipple. The mass is clearly definable; the boundaries are clearly cut and circumscribed; there is absolutely no infiltration into the surrounding tissues, and the axillary glands are neither enlarged nor painful or tender. On endeavoring to move the mass it moves, taking the greater portion of the breast tissue with it.

COMMENTS AND OPERATION

The patient states that she has never traumatized the breast, and has never had any mastitis or any trouble of any kind with either breast.

In examining this patient's breast I was not impressed with the fact that the mass moved alone without taking the rest of the breast tissue with it. There was some fixation of the skin over this tumor, and it was not freely movable, as the history states.

This woman had a goiter, but it was not exophthalmic. She had none of the symptoms of Graves' disease. Three years ago I operated upon her and removed half of the thyroid gland. At the present time the gland is hypertrophied. In the midline there is a large, circumscribed, firm, non-fluctuating mass, which seems like an adenoma of the isthmus of the gland, the disease having returned. Now, there is a very grave reason for asking ourselves the question whether or not this is an adenoma of the isthmus, because of the relationship which this tumor may bear to the tumor in the breast.

Tumors of the thyroid gland either have a point of election of metastasis or they follow a physiologic selection in their metastasis. There is a definite physiologic relation between the thyroid gland and the long bones, and for that reason metastasis occurring from the thyroid gland takes place in the shafts of the long bones more frequently than in any other position, but not exclusively. I am speaking now of malignant lesions of the thyroid gland. If you have a sarcoma in the thyroid gland, you have a metastatic sarcoma more commonly in the upper end of the tibia and in the upper end of the humerus than anywhere else. Then come the bodies of the vertebræ and, less frequently, the other bones. That does not apply to the innocent or benign tumors of the thyroid gland. In these innocent tumors, known as the metastatic variety, you have from the thyroid gland metastases occurring in tissue which is not microscopically differentiated from normal thyroid gland tissue. These metastases do not occur in the long bones, but in the flat bones, and more particularly in the calvaria—the parietal bones; next in the short bones, like the bodies of the vertebræ, and so on. But these are non-malignant metastases of the thyroid gland, so far as we know. When you have tumors of the thyroid gland, they not only metastasize in the bones, but in other structures, and the breast is one of them.

The question is whether this metastatic lesion in the breast is from the tumor of the thyroid gland. It is our belief that the tumor of the thyroid is a circumscribed, non-malignant adenoma; therefore, usually, we should not have metastasis in the breast from that variety of tissue. However, a microscopic examination will settle this point.

The tumor, as it appears to us from the physical findings, feels like a sarcoma or carcinoma. I have said sarcoma because that form of tumor is rare, but sarcoma grows rapidly and, when it occurs in the breast, is much more malignant than carcinoma. It recurs more frequently in the neighborhood, and it is not transmitted through the glandular chains or through the lymph-vessels. It is transmitted and takes its metastasis, in the great majority of cases, from the circulation, and not from the lymphatics; therefore, you find no involvement of the lymphatic glands in the sarcomata of the breast, as you do in the carcinomata. Fixation of the skin occurs as well with sarcoma as with carcinoma, but not so frequently. It is more commonly a circumscribed, encapsulated mass, and, notwithstanding that the capsule separates it from the neighboring structures, it has a tendency to local recurrence, as well as to hematogenous transmission to other positions. I want to impress upon you what I desire to call your attention to, namely, that the skin moves but a short distance, when it immediately commences to pull the tumor with it. You can see that the tumor slides with it. This tumor is irregular in its conformation; it is woody hard, so we operate on the basis that it is a malignant lesion that moves on the chest-wall, and it means that this whole breast must be removed. The tumor involves two-fifths of the lower outer segment of the breast. Please notice the fixation—and that is the best test of fixation as I try to move it. When I lift the skin in the neighborhood it does not move very much. It is fixed to the skin, and this is one of the best means of determining fixation. It draws down through the fixation to the capsule of the tumor.

I make an elliptic incision, cutting from away above the level of the axilla on the pectoral muscle, making an ellipse. We will take out this nest, commencing from above. You will notice that I am going away under the skin, wide of the breast, taking off all the pectoral fat down onto the aponeurosis of the pectoralis major. You see the aponeurosis. In our surgical work in this clinic we do not take out the pectoral muscle: we retain it for the management of the axilla. I have not decided what the treatment of the axilla will be until we remove this tumor, and then we will determine what is to be done. Remember that the metastasis will not take place from the muscle. Metastases do take place in the aponeurosis of the muscle. Remember the metastases follow the lines of lymphatic chains. These run through the aponeurosis and rarely ever through the muscle, and, in the experience of Mr. Bryant, of London, in the Guy's Hospital service, extending over forty years, there were only two metastases, if I remember correctly, occurring in the muscles in that period, showing how rarely metastasis occurs in the pectoral muscle. It occurs in the sheath of the pectoral muscle, and that is why you note here that we have taken off all the sheath of the pectoral muscle with the tumor. We are retaining the pectoral muscle for another purpose—for the protection of the vessels and nerves in the axilla, to avoid compression that occurs and the edema and swelling of the arm that are such common sequelæ following the removal of the axillary structures.

We have removed the breast. We will make an examination of it. There is no doubt about the nature of this disease. It is not encapsulated; it is an infiltrating, diffuse mass, and you see how it contrasts with the description that has been given in the history. There is no question in my mind, from the gross appearance, which is just as characteristic as the microscopic appearance, that it is a carcinoma. We must take out all the axillary glands.

If there is a metastasis from the neoplasm in the thyroid gland, we will very easily determine this with the microscope, because if it is a metastasis from malignant disease of the thyroid gland, it will have the typical thyroid cells in that neoplasm. It is in this that malignant diseases differ from all other kinds of infections in their metastases.

When you have a metastasis in any other type of infection, with the cells that participate in the enlargement and in the new neoplasm at the point of metastasis, there is always the cell of the tissue in which the metastasis takes place.

Why? When it is a malignant lesion, as a sarcoma or a carcinoma, and metastasis takes place, then it always has a type of original cell, and it is this cell that makes up the great bulk of the tumor, and the cells in the new position of growth play only a secondary and unimportant rôle, and they do not multiply or assume the characteristics of malignancy at all; the secondary metastasis and the primary metastasis are exactly the same type as the primary tumor, and do not take on the character of the cells of the primary metastasis. Therein malignant metastasis and malignant disease do not in any way resemble in their clinical course or in their histologic action the infective lesions, and that is what makes me believe, all the time, that it is not an infection. That is the one thing wherein it deviates from all other types of infection. The nearest thing to the production of cells of this type are the cells produced by the injection of scarlet red beneath the epithelial structure. When scarlet red is injected beneath the epithelial structure, it produces epithelioid, if not epithelial, cells, which so closely resemble the non-epithelial cells that they cannot be differentiated from them with the microscope. This is one of the things that leads me to believe that the production of the type of cell and the nest formation of cells that occur in carcinoma are due to some toxic substance and not to a microörganism.

In the production of carcinoma in the breast we have a little different clinical course from that commonly seen in carcinoma in other positions in the body, in this particular: a carcinoma in other positions in the body is usually the sequence of repeated mild traumatisms. When trauma is associated with carcinoma of the breast, it is usually one moderately severe trauma. You see, that is a very great difference. Sarcoma is produced, as a rule, with one moderately severe trauma, not sufficient to produce, for instance, if it be to a bone, a fracture. In carcinoma of the breast it is usually not sufficient to produce a laceration, and I know of no case recorded of carcinoma of the breast that has resulted in the scar of an incision of the breast except where the operation was done for malignancy.

In making this incision you see it is our purpose to keep the line of incision entirely out of the axilla. The skin incision is entirely out of the axilla. We are in the axilla with a superficial incision. The next thing we shall do is to remove all the aponeurosis of the pectoralis major in this zone, and all the fascia and fat that exist between the pectoralis major and minor muscles. It is in this position that we not infrequently find a single lymphatic gland, and it is in this position that we not infrequently have metastasis. The next step in our procedure is to pass the index-finger under the pectoralis major and free it so that we can displace it outward. We split that parallel to the long axis of the fibers; we have the anterior thoracic vessels coming down and supplying this flap with nutrition. See them shining in position! You see I have all the advantages I can get from removing that muscle. I have all the advantages that can be acquired. I am on the pectoralis minor now, and I have the muscle there for a splendid purpose, which you will see later. The next step is to expose the margin of the pectoralis minor.

You see it shining there. I lift that up, and you will see what a splendid exposure I have clear up to the clavicle, so that I can commence now to dig out all the glands and the gland-bearing connective tissue. Notice the intercostal muscles shining beneath my finger. We expose the side of the chest, and beneath we see the axillary vein; we will expose the upper surface as well. You see the shoulder branch of it passing off with the thoracic branch on the inner side. The vessel is nicely in the field, and we will commence to take off all this fat from the thoracic division. These veins have scarcely a plus pressure; the finest ligature suffices. If you tear a vein, do not ligate it, but suture it. On inspiration it has a minus pressure; on expiration it has a plus pressure, so you have nothing to fear from leakage from a suture.

We are back on to the latissimus, as you see here. I show you the long subscapular nerve, and there is the vessel. See them exposed! I wish to retain them, otherwise the woman will not be able to hook her belt behind. There is the latissimus showing in the field. We have taken out the great zone from which the danger of infection exists—that is, metastatic infection, that is around the vessels and deep up to the clavicle. It is the all-important matter in preventing metastasis. After we have removed this portion of the attachment above the vessels we will inspect these glands and see how they appear to us. This gives a perfect picture of the axillary zone; here are the nerves and the trunks of the nerves.

In Greenough's report of the swellings of the arm, if you will remember, which he made before the surgical section of the American Medical Association, analyzing the cases in the Massachusetts General Hospital running over a period of ten years, as I recollect it, he showed that 31 per cent. of the patients had edema of the arm and a little smaller percentage had neuralgia in the arm following operation. The edema of the arm is usually attributed to the lymphatic stasis. I think this is an error. I believe the edema in the arm is due to the compression or resistance to the return circulation by the cicatricial contraction of the tissue that forms in the axilla. You can see, when you analyze that axilla as it is now, there is a great dead space. We must fill that dead space. We provide nothing, when we remove the pectoralis major and minor muscles, to fill that space except new-formed connective tissue which encircles the vein for at least three-fifths, if not four-fifths, of its circumference. It encircles the nerve-trunks in the same manner, but not to so great a degree.

What occurs to new-formed connective tissue in every position in the body? Contraction and compression, and, if it is an annular compression, it may amount to strangulation, as was shown by the results of Schede's operation for varicose veins of the leg, and that an annular scar surrounding any structure produces obliteration of it. In looking over the specimens in the British museums when we were studying arterial suture, we found that the thoracic aorta was completely obliterated through cicatricial contraction from lesions occurring in the mediastinum, from infections from the esophagus, or from diseases of the spinal bodies. Just the same thing occurs here, and it is our belief that it is a cicatricial contraction or a contraction of the new-formed connective tissue around the vessels and nerves in the axilla that produces the pain and the edema, or rather that produces the edema and pain. The edema is a serious matter in many cases. For ten years or more we have adopted this plan: I have removed all the subpectoralis major fascia and intermuscular fascia between the pectoralis major and minor. We have, therefore, removed all the chains of lymphatics through which metastases occur, and we have accomplished as much for the protection of this patient by the removal of these as we can by the removal of the entire pectoralis major muscle. At the same time we have preserved the pectoralis muscle for a definite and important purpose, namely, the protection of the artery, vein, and nerves by implanting this pectoral muscle into the axilla.

You will remember the splendid papers of Carwardine, who showed that if we could diminish the axillary angle by getting skin to go in there, we would have better final results and solution. There is one little area in the center here that was not palpable before the operation, but it is clearly demonstrable now. As I have said, Carwardine has pointed out that if we could lessen the angle and fill in the space we would lessen the tendency to edema. So important is this edema that Handley has had to resort to implantation of tubular silk in the pectoral muscle and in the pectoral fascia, so as to relieve the stasis of lymph which causes the swelling of the arm. He implants the tubular silk there away down here in the arm, through the back, and through this tubular silk he drains off the stasis that occurs. We are implanting the pectoralis major to fill that dead space. The pectoralis muscle has been removed, or as much of it as is necessary for the protection of the patient against the dangers of metastasis. We have as much protection as can occur from the removal of the muscle. This is not only based on anatomic knowledge, but on clinical experience. We have never seen but one return of neoplasm in the pectoral muscle, and never a return in the pectoral muscle implanted in this way, showing what a rare condition it is. You will notice that I catch the latissimus behind. You will see the long subscapular nerve in the field; we will bring that out, and with this stitch turn the end of the muscle away up into the axilla, so that we will have soft muscle surrounding this vein and these nerves, and we will have no dead space that is to be filled with connective tissue which subsequently contracts. Four or five stitches will suffice for the fixation of the muscle. It adds nothing to the gravity of the situation; if anything, it is an aid, as has been demonstrated by Horsley, where muscle is implanted on a bleeding surface. See what a different aspect the axilla presents now! The tissues come up, it is all filled at once, and you have a perfectly movable arm. One good thing about this operation is the freedom of movement of the arm, not only after the operation, but continuously so after it. We have ample skin to cover all this field.

Taking up the lines of metastases as they occur in frequency as well as in number, we have, first, to consider that the chains of lymphatics leading to the axilla represent more than four-fifths of the lymph-channels leading from the breast; that the lymph-channels leading to the skin in the intercostal space represent about one-tenth; that the channels leading to the sixth interspace represent about one-twentieth, and we would estimate, from the frequency with which we see metastases in the liver, that Handley's idea of transmission represents about one-twentieth of the metastases. Handley's idea is, as you know, that carcinoma is transmitted along the lymphatics into the umbilicus and along the round ligament, and in this way the cancer-cells reach the liver. Our knowledge of the lymphatic system is that they do not lead from the round ligament into the liver, but that they lead from the round ligament along up through the mediastinum, and that the danger of hepatic metastasis does not occur from transmission of the cancer-cells through the lymphatics at the umbilicus, but that they are of hematogenous origin, the same as they occur in the long bones. You know that carcinoma of the breast has its most frequent metastasis in the bones after the lymphatics; most frequently again in the long bones—more frequently in the upper end of the femur than any other bone. The second intercostal interspace is one of the common positions for glandular metastasis—the second intercostal space just to the side of the sternum. The recurrences *in loco* in a carcinoma, when the carcinoma is small, are more commonly in the skin than anywhere else; less frequently on the chest-wall, beneath the pectoral attachments, and less frequently in the lymph lines—the radiating lymph lines—from the mammary gland in the various directions that I have mentioned. You will note, from the trend of my conversation, that I believe the removal of the pectoral muscle has little, if any, influence on the metastasis or the return of the disease *in loco*, and that is why I use the pectoral muscle, using either its anterior or its posterior fatty fascia, which also carries lymph-channels. I took off the subpectoralis fascia, which is a part of the general fascia of the axilla, or the fat-bearing part, all of these having been removed.

As to the final results in carcinoma of the breast, they are just as disheartening as are the final results in carcinoma in almost every position in the body except in the fundus or convex surfaces of viscera. The final result of carcinoma is dependent on two avenues—let me make that three, so as to avoid controversy. First, the location of the neoplasm as regards the richness of the lymphatic vessels in the neighborhood in which the carcinoma originates. This is the all-important controlling element. Second, the resistance offered by the patient, which, I believe, is indicated in the condition known as scirrhus. Third, the lesser malignancy possibly of the cells, which I do not believe. I believe that the carcinomatous cells are equally malignant in all individuals, but that the resistance is of two types—the resistance formed by the particular tissue that is involved, and the resistance that is formed by the patient in general. In other words, a scirrhous condition is a manifestation of a reaction of the connective tissue to the irritation of the invading erratic epithelial cells and their toxic products. A patient who has the great resistance offered by a coffer-dam of connective tissue which consolidates and contracts should give us the condition of scirrhus. A patient that has little resistance has his cellular tissue penetrated and infiltrated, and the connective tissue makes no effort at resistance. You find exactly the same lines of resistance running all through the tuberculous lesions. First, there is local tissue resistance, and, second, there is the resistance of the patient in general.

We divide the cancer-cells into two varieties: the squamous-cell variety and the glandular cell or the columnar cell variety. The squamous cell is the most luxuriant in growth; it tends to irritate the tissue *in loco* and to produce encapsulation, as seen in carcinoma of the lip, which is really an epithelioma. An epithelioma of the skin which we not infrequently see in old people, both microscopically and clinically, is practically non-malignant, because we see these people going along ten, fifteen, or even twenty years with the slowest possible growth of this condition, but if they should get some additional irritation, the epithelioma becomes more malignant. It differs entirely from the columnar cell carcinoma. The columnar cell carcinoma, which is present in the lining of all the viscera, as the cervix of the uterus, the pylorus of the stomach, the neck of the gall-bladder, has a much greater tendency to penetration. It has a very much less induction to connective-tissue reaction, and, above all, it goes back to the first element which I accentuated, that the columnar cell lesion is, *par excellence*, the lesion of the cervical portion of all the viscera in which you have the greatest and richest lymphatic supply of any viscus. The rich lymphatic supply of the gall-bladder is in its neck and ducts; the rich lymphatic supply of the stomach is at the pylorus; the rich lymphatic supply of the urinary bladder is at its cervix or *bas-fond*. These are points at which you have columnar cell carcinoma occurring most frequently. Here we have less hope of cure, because the cancer-cells are transmitted through these rich lymphatics so easily after the penetration of the basement membrane by the erratic epithelial cells.

In considering the question of carcinoma of the breast we did not touch on the final results in an accurate way. I believe the final results in carcinoma have not been very materially improved in the last quarter of a century. All you have to do is to look over the statistics of the men who have made the maximum number of operations and analyze the histories of their cases or their reports. You will find, of the cases that have no demonstrable lymphatic metastases at the time of the operation, that these patients remained well in a considerable percentage of the cases, exactly as they did with our forefathers in surgery, who excised breast tumors and did nothing else. When there were demonstrable metastases, you will find there was a sudden drop in the percentage of permanent cures, and in no place is that more striking than in epithelioma of the lip. Epithelioma of the lip, where there is no demonstrable glandular involvement at the time of the operation, has a cancer mortality of 52 per cent. Epithelioma of the lip, where there is a demonstrable glandular enlargement at the time of the primary operation of excision of the lip, has a cancer mortality of 76 per cent. Nearly all of them die. These figures are taken from an analysis of the cases in the London Hospital, where they were gone over very thoroughly. The same could be said if you analyze the statistics of Halsted, of Rodman, and of those who have advocated a very extensive operation in carcinoma of the breast. For instance, we have the Handley operation, where you take the fat off of the anterior wall of the abdomen, cutting off one-twentieth of the line of metastasis—where you take the fat off clear down under the umbilicus and take out a part of the round ligament. The futility of an extensive operation in carcinoma of the breast can be estimated from the percentage of recurrences that take place in the osseous system and the liver, but particularly the osseous system, following excisions of the breast in which there is no demonstrable return in any portion of the breast or glands leading from the breast before the patient exhibits the disease in a bone, or the disease terminates fatally. You can see that these metastases have occurred long before we make the primary removal. If there is any hope to be expected from operations for carcinoma of the breast beyond what we now realize, it is this, that we must recognize the tumors earlier and remove them promptly, and that is based rather on a dream than on positive knowledge except the knowledge of clinical experience. We have no facts, or anything supported by facts, so far as our knowledge of the method of transmission and time of transmission of the erratic epithelial cells to the other structures is concerned. No one has yet shown how an epithelial cell, which is erratic, passes from its erratic position in the subepithelial basement membrane to the next lymphatic or lymph-space. This has not been shown even in mouse cancer. It was expected that Gaylord would show how these early metastases would occur in the mouse. We do know, however, that in carcinoma, scirrhous in type, hard and indurated in its local position, when there is a strong connective-tissue reaction in the primary lesion, there is quite a strong resistance to the metastatic lesions, which makes me believe that this resistance is more constitutional than local, because in every position it is the same type of resistance. You can infer that, and say possibly the cells are less malignant: but if less malignant, why do you have so great a "coffer-damming" of tissue to take care of? It is in those cases that have a firm, strong, resisting fibrous capsule around a nest of epithelial cells that the tumor is of slow growth and slow in metastases.

LIPOMA OF THE SHOULDER

HISTORY

Female; age sixty; entered hospital to have a growth removed from the left shoulder. Family history, father died from cancer of the stomach at the age of fifty-three; mother, of pneumonia, at the age of seventy-nine. Past history, ordinary diseases of childhood. Sixteen years ago had a vaginal hysterectomy performed at this hospital. In September, 1909, was ill with an infection originating from a cut over the tibia from a fall against the curb. Personal habits good. Married forty-two years. Menstruation was always normal preceding hysterectomy. Venereal, negative. Has had five children, and three miscarriages following the birth of the first child.

Present Trouble.—Patient noticed, in 1903, a slight enlargement on the top of the left shoulder portion of deltoid. It has not grown much since then. Two months ago the shoulder began to ache, and up to the present date has continued to cause discomfort, in the form of a dull ache, at variable intervals. She has no glandular involvement. No tenderness on pressure.

COMMENTS AND OPERATION

I am going to make this incision lower than usual, so that the carrying of weight on the shoulder subsequently will not come on the scar tissue. It comes far below the line of pressure—the weight-bearing position. This is a cancellated lipoma with many diverticula. You see the diverticula as they pop out in various places and close under the skin. This tumor originates from the fascia, which is the common point of origin of this type of tumor, and from the aponeurosis of the subscapularis muscle. It is essential that all the basal portion of its origin should be removed, otherwise the tumor will recur. When it is the type of lipoma that appears in the fatty tissue itself and does not originate from the aponeurosis of a muscle, then the tumor is not so likely to recur, and you do not have to remove the aponeurosis of the muscle, which I have done here.

There is no plase where this is so striking as in operations for the removal of lipomata that occur along the spinous processes. You have a group of lipomata in which the tumors most commonly occur in the lumbar zone, less frequently in the lower dorsal zone, and the next in frequency are those which occur in the lower cervical zone, then the upper dorsal zone, and, finally, in the sacral zone. These lipomata spring, in a large percentage of the cases, from the periosteum, practically never from the body of the vertebræ, but always from the processes, either the laminæ or spinous processes or transverse processes. They are usually forced through when they occur on the spinous processes, going between the erector spinæ muscles, and are forced from one side to the other, and appear often on the opposite side from which they originate, passing through the membrane which connected the spinous processes. Those that are operated on and cut off at that septum always recur and continue to recur until such time as you go in there and take off the periosteum from which they originate.

There are two classes of tumors that occur on the posterior surface of the body in the midline that are deceiving in their appearances, and unless their embryology or origin is appreciated, they always recur. One is the postsacral type of dermoid, great or small, and the other the lipomata of the spinous processes. In the postsacral dermoid, unless you excise all the tissue, the tumor will recur. In the spinous processes, where you have lipoma to contend with, unless you excise the periosteum from which it originates it will continue to recur, and it is often diagnosed as sarcoma because it recurs when it is only a simple periosteal lipoma.

In the treatment of the postsacral dermoids there is no amount of cauterization, or tickling, or trituration, or touching, or whittling of pieces of it that benefits in the least. It is an embryonal structure of epithelial origin, and is cured only by complete excision, and complete excision is as often a difficult task with the postsacral dermoid as it is with lipoma of the spinous processes. Once they begin to suppurate or irritate, removal is the only method of cure. Removal by periosteal excision is the only method of final cure of the mesoblastic product.

VARICOCELE

HISTORY

Male, age twenty-four, by occupation a grocer, was admitted to hospital because of a dragging pain in the scrotum, most noticeable on the left side. Family history, negative. Past history, has never been ill since childhood—as a child he had measles. He smokes five cigars a day, but uses no tea or coffee. Absolutely denies any venereal infection.

Present Trouble.—Started four years ago, 1908; the patient states that at that time he noticed a slight enlargement in the left scrotum. Since June, 1910, he has had a dragging sensation in the scrotum; this sensation has occurred at variable intervals for the past year. The discomfort becomes more marked when he stands or walks for any distance. Examination shows a number of worm-like processes in the left scrotum.

COMMENTS AND OPERATION

The history, as you have heard it read, is typical of varicocele, which is an innocent lesion when it is not disturbing the patient, but is a very annoying lesion when it does. When a patient begins to get backache and a dragging sensation from a varicocele, then it is important to the patient.

In the last one or two decads we have got so into the habit of considering the tendency to fatal termination of a lesion, and overlooking the clinical or symptomatic and disturbing elements of the lesion, that we have forgotten the patient almost entirely. In other words, the school of pathology did not carry along with it the desire and the training for the relief of the symptoms of the patient as it progressed in its efforts to relieve him of the pathologic condition. It is hardly necessary for me to say to you that patients come to us for the relief of symptoms, for the relief of pain, discomfort, and annoyance, and a patient from whom you have removed a carcinoma of the pylorus is not so grateful to you for the result as is the man who has had an ingrowing toe-nail successfully removed. This is an absolute truth, and I think in that particular we have been negligent in considering the comfort of our patients both before and after operation. There are so many ills that have no gross demonstrable pathologic manifestations that when we inspect the region in which the pain is located, we are not only likely to say, but we do say, that this is a neurosis; or we call it neurasthenia, the definition of which is incompetency on the part of the doctor to make a correct diagnosis. This is the right definition of neurasthenia, and I speak of it because we so commonly and uniformly overlook the etiology of the symptoms producing the lesion. In other words, they are not associated with gross pathologic lesions, and this is just one of them.

This man has a dragging sensation in his scrotum. He has pain in his back. He has discomfort, and when night comes he has one ache in his scrotum until the morning, and to him it is just as important to relieve that pain as it would be to relieve the pain and discomfort which are always less in a case of sarcoma of the testicle and elsewhere, and in handling these cases we must be more considerate for the patient's ills and pains and aches. In my mind, a lack of this consideration is the reason why all the cults of medicine have originated. I believe this, that the regular family doctor is always the first choice, and it is only after his shortcomings have been grossly accentuated by his inefficiency that anybody else is called upon to afford relief. The next decad is going to bring a constant and forceful current on the relief of the symptoms of a patient, as well as relief from the gross pathologic entity which in some cases is discernible, but in a large percentage of cases is not.

You will notice that these veins almost entirely disappear when the patient is in the recumbent position, and are not clearly demonstrable. In the last two cases operated on previous to this one we had the opportunity of showing you how these veins could be dilated by continuous compression just below Poupart's ligament and across the cord and mesentery. By holding the pressure on continuously, the vessels would enlarge and enlarge. We make our incision behind and fairly well forward through the scrotum. We have, first, dilatation of the external veins of the pampiniform plexus, as you can see. We will be able to demonstrate these veins when they commence to show up with just a little pressure. As I make a little pressure you will see these veins standing out here in nests. It is this nest that it is so important to locate and remove. It is the dilatation of these veins that produces the severe backache; it is the dilatation of these veins that produces many of the reflexes which result in the various sexual diversions that are resorted to by so many young men. You will find very often that it is the result of dilatation of these veins that emissions are kept up, because you have rendered the spinal center from this zone very irritable from the constant irritation. Notice the size of this vein—it is almost as large as my little finger. Here it anastomoses with a bunch below, and here we have to exercise care in its removal, as it is down onto the epididymis. Note it going around. You will see by the spreading dissection how easy it is that it works out. The central veins along the spermatic cord are not involved, and they do not need to be excised. We are tying all these veins below. We draw them off just a little above a plus pressure. We keep the ligatures all long, suspending the testicle after the completion of the operation.

Backache is so common a symptom with people that we do not give it sufficient attention, nor do we go into it sufficiently deep to make an accurate diagnosis of the etiologic factors of the backache, to use that expression, or the etiologic factors in the production of the backache. The great bulk of these patients come to the office and complain of lumbago, and we accept the term lumbago because it sounds more than a backache. But it means the same thing. It means exactly the same thing, so far as our treatment is concerned, and so far as our knowledge of the factors producing it are concerned, namely, that we are satisfied to tell these patients they have rheumatism; or we call it lumbago, when they call it backache, and they go home thinking they have learned something, when the fact is that it is the same thing.

You will notice that I am tying the upper stump of the vessels to the lower just in one knot, and that will hold it suspended so that we can get organic union of the structures, and it will not descend and continue to give a dragging sensation in the future, and the patient will not have pressure, either hydrostatic or hydraulic, produced on these veins.

As a rule, when a patient comes to us with a backache, the backache has a definite pathologic origin, particularly when the patient comes to us at this season of the year. In my practice the backache of springtime is different from the backache in the fall. We have a large out-of-town practice,—in fact, 82 per cent. of our cases come from out of the city,—and a large number of those who come have some trouble with the spine. They have not been educated to the "cheiro-practic" idea. When they are, we will not get them any more unless we do better work for them in the future than we have done in the past. Many of these patients come in from the rural districts, some from cities, and some from towns. They have been handling various farm products during the fall or the active season.

There is a typical fall epidemic of backache, which is either positional or traumatic spondylitis. One man states that he believes that his backache was produced by carrying barrels of apples; another man says that his backache has been caused by carrying large quantities of grain; still another man comes in and says that he has been husking corn or digging potatoes and believes his backache is due to that. But in each individual case that comes in you can find a definite etiologic factor absolutely, just exactly as in the coal districts of England, where the miners have a change in the conformation of the sacral or lumbar joints because they always work in this stooping position, and they have what is known in those districts as coal-miner's backache, just the same as our people have it who work on the surface in the particular season of the year, and not infrequently it is a grave lesion where it is associated with a fall cold or a fall grip. You then have not alone pain, but a metastatic condition, and you have an ossifying spondylitis, starting in the back and lumbar region and going right straight up the spine until every spinal body is fixed. I have in my office a specimen of that type, where everything except the atlas is bound to the sternum from that type of metastatic infection. The milder forms that are not associated with infection are curable by the application of a plaster cast, leaving it on for ten weeks, and, immobilizing the articulations, you have the disease subside, but it returns the next season if the patients are exposed to the same cause. But this metastatic deposit that has taken place is permanent and leads to permanent fixation of the spine.

What are the ways by which we can demonstrate the presence of this disease? In the first place, the patient should be stripped. You know as well as I do that the average patient with a backache, when he consults a doctor gets a prescription, and not an inspection. If you will strip the patient and look at his feet, and have him fall back on his heels with his legs straight together, you can see the spine is fixed. It is immobilized in anterior and posterior motion more than it is in lateral motion. In these cases of spondylitis there may be complete obliteration of the cartilages and more or less joint destruction. It is important that one make an inspection and examination of the back in every one of these cases, to find out why the patient has backache, and then prescribe for it or order something that will do him some real good. If it is a traumatic spondylitis or a positional spondylitis, give him a sole-leather jacket or a plaster-of-Paris jacket, and in a short time you will be surprised to learn how good a friend you have made of that patient. He will tell you that he has taken all kinds of prescriptions, the salicylates and other things of that type, and that they have not benefited him. But if you immobilize the spine, you will stop the whole process.

If he has a metastatic deposit and you recognize it, with the presence of inflammation, in some other part of the body, you have a bad case. Yesterday I saw a little child eleven months of age. The first doctor who examined the child resolved that it had a dislocation, and attempted to reduce it. The child had no history of anything to produce a dislocation. Nobody had handled it severely; it had no crying spells and no evidence of injury, but the right arm was immobilized, and it could only move the right arm with the left. The next doctor, who looked the little patient over, saw that the joint was swollen and was perceptibly 30 per cent. larger than the other joint, and said that the child had rheumatism. The mother told the story without any solicitation. I said to the mother, "I want you to tell me what you first noticed wrong with this child." She said, at first the child had a cold, which dragged along for about ten days, and then one night it was taken sick and began to cry. The next morning it had a fever; the temperature rose to 104° F., and had continued from that point to 101° F. ever since. Any one who knows anything about dislocations of the shoulder knows that this is not the clinical history of a dislocation of the shoulder. The shoulder was thickened, edematous on the outside, and examination by the *x*-ray showed a mass filling the sac, but the epiphysis of the humerus was still intact. The fact that the epiphysis is yet attached makes me feel, considering that the child is only eleven months old, that it has a primary metastatic joint lesion. The metastatic disease from the cold which the child had was immediately followed with a metastasis, this occurring ten days after the onset of the cold, the chill, the restless night, and the high temperature, although no temperature was noticeable preceding the metastasis. That is a uniform typical history. It is one of the things that occurs every day. Do you wonder that parents are dissatisfied? I had to tell both the doctor and the parents the facts in the case, because I felt that the child was going to have an ankylosis of its shoulder, and that it was essential for me to tell them now, before I assumed responsibility for the management of the case. Four weeks have elapsed since the metastatic infection. If only four days had elapsed, this child would not have ankylosis. In this connection I recall one of my very early experiences with the late Dr. Fenger. The child had scarlatina, and following that attack had immobilization of the hip. The child was then two or three years of age. Dr. Fenger said that this was a metastatic inflammation of the hip from the scarlet fever, and he opened the hip on *that* day, not the next day, put in a skin drain, and the child did not have a particle of trouble with the hip. This was done over twenty years ago. This case showed the importance of relieving the tension of the products of infection in a joint and not draining the joint too much.

NERVE ANASTOMOSIS

HISTORY

Male; age, forty-one. Works in roller mill. Admitted December 4, 1911.

Was admitted to the hospital because of a paralysis of the hand, wrist, and forearm, resulting from a cut across the antecubital space, about two inches superior to the insertion of the biceps tendon. The biceps tendon was severed, and his ability to flex his forearm upon the upper results from the activity of his brachialis anticus. The accident occurred in July, 1909, and resulted from the dropping of a sharp, heavy piece of iron upon the site traumatized. The paralysis of the hand, wrist, and forearm resulted immediately following the injury; the paralysis is absolute. He is unable to flex or extend the fingers or wrist. His skin sensation is entirely absent over the palmar and dorsal surface of the hand and lower portion of the wrist. However, a deep pressure or pinching of the finger elicits a tingling sensation on the upper flexor surface of the forearm, just below the bend of the elbow; likewise a tingling sensation in the palm results from an irritation of the upper flexor cutaneous surface of the forearm. The sensation of the forearm above the wrist is normal. His radial and ulnar pulse is barely perceptible, and feels as though it were due to a collateral circulation or constriction at the elbow. The radial and ulnar pulse on the left wrist, the one traumatized, is not synchronous with the pulse on the right wrist, but occurs slightly after it. The hand is always cold unless it is kept wrapped up. The forearm and hand muscles are atrophied from disuse. He has been operated upon twice before. Examination shows the presence of a neuroma, the proximal end of which is felt about one inch above the scar.

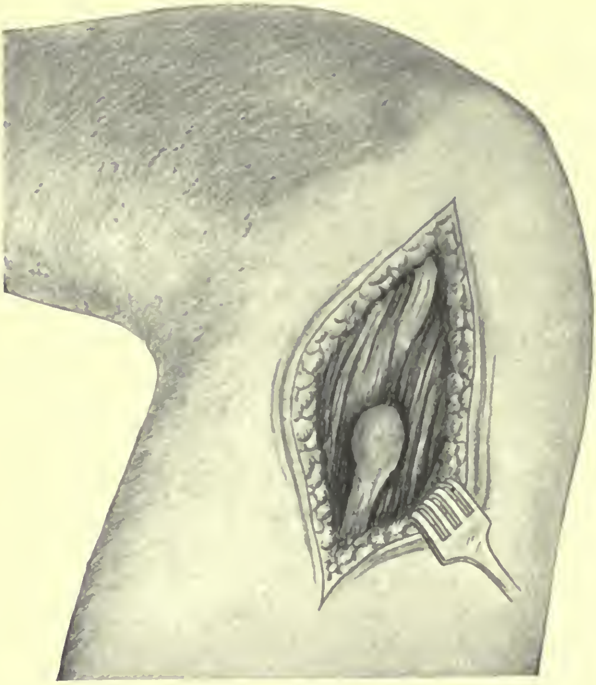

Fig. 1.—Neuroma on proximal end of median nerve.

COMMENTS AND OPERATION

This patient had a primary suppuration to start with, was operated on twice for the restoration of function, and we have the anatomic landmarks entirely obliterated. When we examined the arm yesterday we thought we felt the ulnar nerve. The ulnar nerve is one of the most important nerves.

Every muscle is out of commission. He has three nerves divided—the ulnar, median, and the continuation of the musculospiral—call it the radial or whatever you please. If we could get his ulnar nerve restored, we would have the flexor carpus in commission; we would have two or three of the profundi in commission, and we would have the outer three lumbricales and the three muscles of the thumb in commission. So you can see how much we would be able to do. In rotating the shoulder we are at a decided advantage in handling the ulnar nerve. There is the distal neuroma. I see the position of the neuroma from the outside. I do not feel it now, but I hope to feel it again.

You see the distal portion of the nerve. Here is the distal end of the ulnar nerve. There appears to be something rotating under my finger that feels like a neuroma on the proximal end. Here is the blunt end of the distal portion. Here I take this to be the proximal portion of the nerve. It is not exposed as yet. There appears to be a neuroma on the distal end. It shows very clearly. There is the encapsulation I talked to you about yesterday, and there, you see, is the other end of the nerve, quite a long distance away. The scar covers that field entirely. We here have a typical neuroma of the proximal side.

You remember, many of the earlier operators united that end into the distal, and the result was failure of union because, when the distal end or proximal end, and particularly the proximal end, is insulated by a mass of connective tissue, it produces a so-called neuroma. At right angles we can make the ends of the nerve reach, but there is nothing recognizable as a nerve—nothing but a mass of connective tissue. We are beginning to see some of the brush-ends sticking out. Notice the little lobulated fasciculi as they show themselves. They are the brush-ends of the axons. There are some neuromata. We will have to remove a small portion. I want sections made of these neuromata. The small neuromata are from the distal end of the nerve. Notice the brush-ends of the axons stick out as I push back the capsule. We cannot put these together until we get the ends in position. The ulnar nerve stands stretching better than the other nerves, but they all paralyze, more or less, from stretching. I want to see living fibers in the distal end. The moment you touch a muscle that has live fibers it responds, but there is no response from the faradic current when the nerve is dead. You notice the part I transplant is a living axon, and not a dead one. Remember also that each of the trunks has a very much larger number of axons than is absolutely necessary for transmission. I make use of the perineuron fibrous tissue on the outside to hold this nerve during the process of regeneration. See how secure that is at the point of union! I have secured it at three different positions. In making a bed for the nerve we guard against the interposition of connective tissue. We have filled in the gap. It is difficult to get the musculospiral above and below, as it comes around in the position I show you; then it goes in and divides into the posterior interossei and radial. It was difficult at first to find the other end of the nerve, but we have found it. I think we can stand that elongation. We must keep the arm flexed all the time during the process of repair, so that it will have a good long period of rest. It would not do to put it up in extension, because with extension there would be separation. We will tie off the veins and then remove the constrictor. There is a span of about an inch between the ends of the musculospiral, and this span can be overcome readily by flexing the forearm. I am using the neuron as a bridge to carry and support the nerve at the point of union. Now we have to deal with the anastomotic vein. There is practically no arterial bleeding. These anastomoses of the elbow are more extensive than the normal relationship because we have here compensation for the

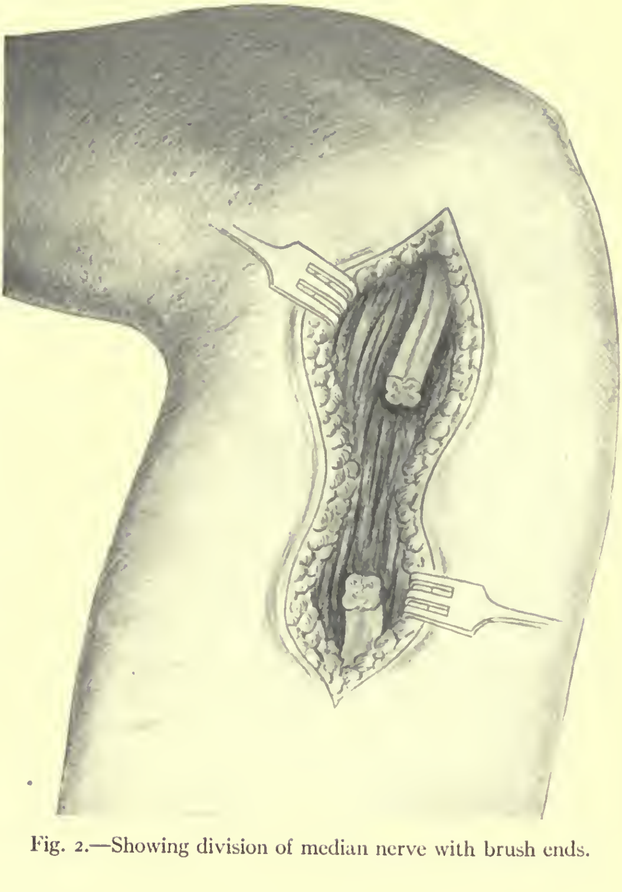

Fig. 2.—Showing division of median nerve with brush ends.

anterior circulation. You see the artery and vein we have divided. We have here each nerve except the ulnar, one spliced by extension and two without extension, although we can bring the other together without an extension and we can contact the musculospiral nerve without extension.

Considering these nerves, how capable are they of regeneration? When we bear in mind that the nervous system is made up essentially of neurons, that the neuron is the unit of the nervous system, you can commence to make all calculations on this basis. There is some question as to whether or not the neuron theory is correct, but that is a matter of little consequence to the surgeon, because the neuron theory fits accurately all our surgical work. From a surgical standpoint, it fulfils everything we need in the analysis of cases of nerve injury, and if the neurologist and physiologist are still in doubt as to the neuron theory, it does not concern us very much as surgeons.

The basis of the neuron theory is the neuron. The neuron is made up of a central cell-body with a nucleus. Attached to it are the long extensions which are called the axons. The short extensions which go to the central cell-body are called the telodendrites, and the finer short extensions that go into the muscle-plates are called the telodendrons. Any part of that cell that is shut off from the central nuclear portion dies. That is a biologic law. Therefore, if you cut out the telodendrons or cut the axon, the distal portion always dies. If you divide a nerve-trunk, you are dividing the axon of that nerve. The axis-cylinder, which is the transmitting portion of the nerve, is an elongation of the nerve-cell substance itself, and when the distal portion is separated from the peripheral portion, the peripheral portion always dies. When that is cut, the axis-cylinder portion that is distal dies because it is an elongation of the cell-substance itself. The medullary sheath crumbles and goes to pieces as it surrounds the dead axis-cylinder.

We have taken the tension off of the nerves entirely by the splicing of an elongation of the neuron, and now we suture the nerve-endings. I have prevented traction from the proximal side by fixing the neurilemma to the fascia of the forearm, so that the least traction will come now on the nerve. Immediately after the division of an axon its distal portion, with the exception of the elliptic cell of the neurilemma, dies. Those of you who are near enough can see this union. There is very little or no traction. You see the support I have used to carry the weight of that and to put traction on when the arm is extended later. Next we shall close the tissue around this line of union.

As soon as these axons are divided, death begins in the central axis-cylinder, then in the medullary sheath, and later you have multiplication of the cells of the neurilemma. They do not die except in their distal ends, and death is due to the irritation of the dead axon and axis-cylinder and medullary sheath. Centrally, with the act of death there comes the act of regeneration, and regeneration is entirely due to the neurilemma.

All the spinal and cerebral nerves, except the nerves of special sense, possess a neurilemma and are capable of regeneration. The optic nerve, the olfactory nerve, and the other nerves of special sense are incapable of regeneration, and when they are once divided, it means they are permanently out of commission, no matter how accurately they are approximated. When the optic nerve is once divided, it never regenerates. In the regeneration of these peripheral nerves you have the rôle of regeneration planned by the neurilemma, producing a connecting link of neurilemma cells from the point of division to the most distal portion of the nerve. It produces a cicatricial or embryonal medullary sheath, and it produces a deposit in that medullary sheath which is appropriated by the axis-cylinder of the nerve when it is once connected with the proximal side; that is to say, there is a tube prepared, within eighteen days after division, in the peripheral portion of every divided nerve for the transmission of the axis-cylinder when the nerve is reunited to a living axis-cylinder on the proximal side. It does not need to be an axis-cylinder of the same nerve, but of any nerve. But it must contact end to end with the stump of the axis-cylinder that has regenerated in the periphery. In other words, there is an active regeneration that is performed in every

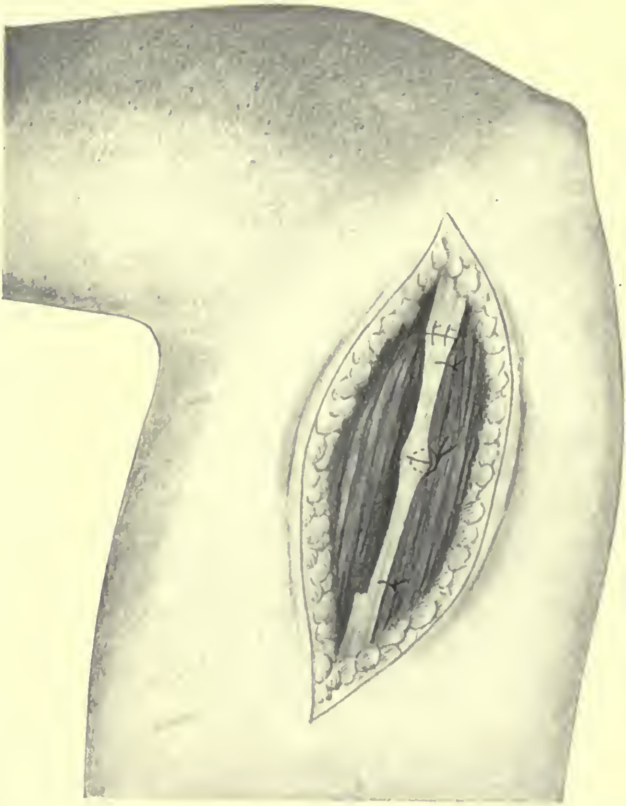

Fig. 3.—Showing median nerve stretched and ends sutured together.

nerve immediately after its division that meets the normal tissue type of repair, but this act of regeneration does not apply to the axis-cylinder, but to the neurilemma and medullary sheath of the axon. Finally, if it is approximated to a proximal living axon, no matter how long after the division, it again regenerates if there is no interposition of connective tissue.

The length of time it will take a nerve to regenerate after it has been stretched or separated will be from six to eighteen months, depending upon how much peripheral connective tissue intervenes between the ends. Here in this median nerve we have to regenerate a large tract of tissue in a transposed nerve, and that will take considerably longer than on the ulnar side, where we have end-to-end union without tension.

This man has had paralysis two and a half years. That is not too long a period from which to expect complete regeneration. We feel that the length of time cuts but little figure in the matter of regeneration. A great length of time can elapse between the time of division of a nerve and the time of its regeneration if one can get favorable conditions for the regeneration. During the period the nerves are out of commission there is muscular atrophy going on. There is atrophy of the muscle-cells. We never have a complete degeneration of the muscle-cells, that is, degeneration beyond the potency of the nerve-cell to restore itself. The muscle-plates diminish greatly in size, but as soon as they are spliced again with a living nerve, they regain their tone and strength.

The reason there has been such a disparity in results in connection with united nerves is that the axonal ends have not been properly exposed. If they had been properly exposed in all the cases, every surgeon would have reported a success if he had united the nerve in a proper way and without tension and without infection.

I see the other end of this nerve. We will pull it forward and close up the anterior gap. We need to splice this as we did the other. We had an interesting specimen a short time ago. We divided the sciatic nerve and turned the ends in the opposite direction. Later union occurred and sensation returned. That is not common in nerve-trunks. In this case that would not occur.

We have elongated the ulnar nerve two and one-half inches, with no tension whatever. There will be no tension on it, so that it needs nothing more than a mere fixation. We dress the wound at right angles with the elbow elevated. We have obtained a much better result here than we had anticipated. I was a little fearful that I would not be able to bring the distal ends of the musculospiral nerve together. I thought I would be able to get the distal ends of the other two, because these two have fairly good trunks below the elbow. At the point where the musculospiral comes out from the interosseous and radial branches we have great difficulty in finding it. It is small, and, fortunately, it came out just right, and we were able to ligate the distal portion without any special trouble. After the nerves are elongated and approximated, a single flap drain is applied external to the ulnar nerve. If it were not for the months we have to wait for regeneration to take place, nerve work would be satisfactory; but both the patient and the surgeon become impatient.

SALVARSAN

COMMENTS AND INJECTION

THE next case is one of secondary specific lesion following a preputial (initial) lesion. This young man was put on mercury early after his infection, and that has delayed the secondary eruption, as it commonly does. Very often the secondary eruption does not appear at all. If you discontinue the iodids and mercury in the fourth or fifth month after the initial lesion, unless you have been crowding them strongly, the secondary lesion will come out. The secondary skin lesions are present in practically all the external cutaneous infections or all the mixed infections that have a submucosa, but in the infection of mucous membranes that have no submucosa, as the male urethra, and in the infections that occur through the placenta, we many times never have a secondary lesion, and the patient comes with a tertiary lesion. These are the most trying cases to handle many years after they have had the primary lesion in the urethra, as a chancre, or have had simply an infection through the placenta. These patients come to us with the central nervous system involved, with the frank statement that they never had a primary lesion. They are honest in making such a statement, and it is true. They will tell us they have never had the secondary lesions, and that is also true, because the spirochetes, in coming from the chancre across on the mucosa of the urethra, arrive directly in the blood in the corpus spongiosum. The spirochetes that are transmitted from the male arrive at the placental membrane and again go directly into the blood. So you see in these two conditions we have a specific infection arriving in the blood without going through the lymphatics, and when it does not go through the lymphatics, often the secondary lesions are entirely absent, that is, the skin eruption, the sore mouth, the sore fauces, ulceration, falling of the hair, etc.

We have in this hospital now two of these cases with a positive Wassermann reaction in both. One of these patients has no other evidence of syphilis than a painless fracture of the hip, but the other has a painless fracture of the ankle, with a typical Charcot joint. The latter has all the tertiary lesions of syphilis of the central nervous system, with tabes. He has only a slight tabetic walk; he has absence of the reflexes; he has the Argyll-Robertson pupil, and he has the Romberg sign pronounced. The other patient was not suspected as having syphilis until he made the statement that he had a painless fracture, and this fact called our attention to the possibility of syphilis; a Wassermann test was made, and in both of these cases proved positive. In the patient with a painless fracture of the hip it was a triple plus, so that there was no question about it. We have in this hospital today a man who positively denied that he had ever had urethral, preputial, or mouth infection; still he has a positive Wassermann reaction, not a double plus or a triple plus, and, as we have some doubt, we are going to make additional tests.

This patient receives 6 decigrams of salvarsan. Salvarsan has not fulfiled its expectations. It was supposed and believed by those who advocated its early use that one injection would bring about a cure. That is already entirely out of consideration, because in a large percentage of the cases there has been a return of the symptoms within a comparatively short time. The question arises now, will it eventually cure if we keep on repeating the injections? We are repeating the injections every six weeks, and following this up until we get a negative Wassermann. A negative Wassermann, however, does not mean a cure of the syphilis, because we get it after giving arsenical preparations or salvarsan, and we get a negative Wassermann after we have given mercury. When we stop giving both, we get a positive Wassermann again. It simply means that we have neutralized that which gives us the Wassermann reaction only. That is what it means. It does not mean that the disease has been cured.

Just a word as to the manner in which we administer salvarsan. In the skin condition we give salvarsan; we give these patients hypodermics of bichlorid of mercury in the muscles, and another plan we have adopted is this: two weeks after starting to give salvarsan we give these patients sodium cacodylate. In the acute chancroidal lesions we give them sodium cacodylate because it cures more quickly than either mercury or salvarsan. We give it in two- or three-grain doses hypodermically once a day, and in eight or ten days the chancre will have entirely healed. After the chancre has healed we give them salvarsan, because it has a more prolonged inhibitory effect than sodium cacodylate.

This young man had his primary lesion about six weeks ago. At that time he had skin eruptions, but under recent treatment these have subsided. The time for the eruption after treatment is from five to five and one-half weeks, and the eruption comes out just as regularly in from five to five and one-half weeks as does measles on the fourth day, if it is not interfered with by the administration of mercury. You will observe that I am making this injection outside of the aponeurosis, not in the muscle, and am injecting it slowly. It produces a burning sensation that is a little disagreeable to the patient. We put a minus pressure as soon as we have inserted the needle, and draw the fatty tissue into the canal, otherwise the salvarsan may run back to the skin and cause a sinus. This patient will be kept perfectly quiet and in bed for the next two or three days. If he has considerable pain, we shall order the hot-water bag, which generally affords relief. We have not resorted to the venous injection yet. Formerly, the salvarsan was neutralized with an alkaline solution, and in some instances it produced necrosis, but since we have been using 40 c.c. of albolene with 6 decigrams of salvarsan necrosis has not occurred. We introduce 3 decigrams of salvarsan on each side. We are not giving it in the veins nor in any other liquid except albolene.

The results from the use of salvarsan from a mortality standpoint are commencing to alarm us somewhat. I have learned of four cases of deaths in Chicago. Two of these followed the intravenous injection of salvarsan, and in two it was injected into the muscles. One of the patients died within a few hours after subcutaneous injection, and the other died within a couple of days. The patient into whom the salvarsan was injected intravenously died on the table, and another patient died shortly after leaving the table.

No surgeon injects anything into a vein without very serious consideration. The inside of a vein is a place that the surgeon greatly respects. The man who is not familiar with the surgical art and with the pathologic conditions of veins will resort to intravenous injections much more readily than would the surgeon. We have been fortunate in not having had an unfavorable result, and I feel like kicking a post when I say this, because we are powerless to control, so far as we know, the conditions that produce the unfavorable results. What they are, nobody has determined. It is not, so far as we know, anaphylaxis. Anaphylaxis always produces death on the table, and always within a short time after the administration of salvarsan or serum.

Some patients cannot stand the administration of antidiphtheric serum. There are people who have horse asthma. There are a lot of people whom we have been calling neurotics because we did not know any better. We say they have a neurosis. Such people, when they get near a horse or ride behind a horse get an attack of asthma. We call it a neurosis, but it is no more a neurosis than carcinoma is a neurosis. There is something in this we do not clearly understand. When a certain patient goes near a horse it produces blood and central nervous conditions which are serious, and if you give antidiphtheric serum to such a patient, whether it be for the cure of asthma or diphtheria, she or he, as the case may be, dies promptly, and it is a calamity. I remember ordering, not very long ago, for a patient who was suffering with asthma, 4000 units of diphtheria serum, and the woman was dead inside of three minutes. Her brother-in-law was a physician. That was an awful calamity. The way in which they die is from edema of the glottis. They immediately become cyanotic; they get edema of the glottis, the air is shut off, and they are soon dead.

Sometimes there is edema of the lungs, and it affects the glottis only slightly. But this occurs in a class of patients who have asthmatic attacks when they get near a horse. I know of one case where a woman could not ride in a buggy behind a horse without having asthma. These patients attribute the asthma to the cold wind to which they are exposed in riding, when the real fact is that it is an anaphylactic condition, and the fatality in this case resulted because horse serum was administered. These patients not only die from the administration of 4000 units of antidiphtheric serum, but they die from the administration of antitetanic serum, the dose of which is small in bulk as compared with the antidiphtheric serum.

CYSTADENOMA OF THE BREAST

HISTORY

Female; occupation, housewife; age, thirty-seven; admitted December 8, 1911.

Patient was operated on in November, 1910, for an adenoma of the left breast, and has returned because of a growth in the right breast, which she first noticed in November, 1910. The first growth which was removed has not returned. The growth is nodular, of the size and consistence of a crab-apple, and is freely movable in the breast tissue. It is located two inches superior and internal to the right nipple. There is also another very small flat nodule, about the circumference of a nickel. This is three inches superior to the horizontal nipple-line and two inches to the right of the gladiolus in the right breast. This nodule is also freely movable, and of the same consistence as the other. Both subcutaneous. The skin is freely movable over both of these nodules; there is no retraction of the nipple, dimpling of the skin, or discharge from the nipple. There is no apparent glandular enlargement in the axilla and no tenderness. The nodule near the sternum gives rise to pain which radiates from left to right along the intercostal nerve. The larger growth also pains, and they both ache at intervals. Both are slightly tender on palpation.

COMMENTS AND OPERATION

This is a classic case in its clinical history. One of the peculiarities of cystic development of the breast is that you have a cyst of similar size and shape developing in the other breast, and, as a rule, it develops in about the same segment. This is the peculiarity of it. Why this should occur no one has explained. These are considered retention cysts, and in a small percentage of the cases of cystadenomata you have occlusion of one of the ducts, retention, and dilatation. Why these things should occur in the other breast at the same site as in the first one nobody knows. When such a patient comes to you for treatment, you should always examine the other breast, and, if you find two similar masses, one in each breast, you know at once, in all human probability, that these are not carcinomata, but cysts. In addition to that, you know you have the physical signs of cyst, which differ so much from the physical signs of cancer. In these cases you have a mass, usually round, sometimes with diverticula or dilatation close to the skin, but in a considerable percentage of the cases on the under surface of the breast. It never becomes attached to the skin except after these cysts have become infected; then they become fixed to the skin and become attached, and this condition causes one to suspect a carcinoma; but when these cysts are found in both breasts, this fact of itself leads you to the conclusion that it is not carcinoma.

One of the things the profession should be cautioned about, it seems to me, is as to pronouncing these lesions malignant to a patient until they have some real reasons for so doing. The patients become so panicky and so frightened at the idea that these cysts are malignant that they are almost unnerved, and very often they will not submit to an operation, but consult some one not in the regular profession.

There is one, as you see. Either one is a small mass, one being below the first. I had difficulty in finding it without the assistance of the patient. It is just on the border of the breast. It was possibly more painful than the larger one, and it appears to be a fibrous mass or a neuroma. In making a division for the exposures of these cysts we always cut in a line radiating from the breast unless we decide to go beneath. These are situated on the surface, and we will not need to go beneath; we will, therefore, cut down onto the capsule of the tumor in a line radiating from the breast. I have decided not to open this cyst until I demonstrate it to you. You can see a bluish tumor shining through. You can see it nicely. I am endeavoring not to open it until I demonstrate it to you. It is well up on the surface of the breast. Now it is opened. These cysts are always of larger size than one suspects from an external examination. They are lined with epithelium, and the method of their removal is the one you see here, namely, enucleation of the sac. Remember, I said they were epithelial lined. This means that you must enucleate the sac if you expect to bring about a permanent cure. Unless you remove all this cyst-wall, which is epithelial lined, there is danger of recurrence. Knowing the pathology of these cysts, you know just about the line of procedure you have to resort to for their removal. As a rule, the cyst is never attached below. It is a retention cyst. There is the muscular base from which it is peeled off. We use these sutures to close the surface, so as to obliterate the dead space and prevent the formation of a hematoma.

In making a diagnosis of probable malignancy in such a case as this, aside from the fright you give the patient, you not infrequently drive the patient away. The patient goes to some one who puts on a paste, and in some cases apparently cures the disease and receives due credit for curing cancer. It is essential for you to examine such a case very carefully and then base your prognosis on your diagnosis. By so doing you will gain the confidence of the patient, and that patient will do just as you advise and will feel safe. A large percentage of the cases of cancer said to be cured by the application of paste years and years after are cures of conditions that were not carcinomatous, but were pronounced carcinomata, not only by members of the regular profession, but by the irregular profession, and are cured permanently by members of the irregular profession, who receive credit for these cures of so-called malignancy by the application of paste.

Occasionally there is a recurrence in the same breast. It is a peculiar thing, but I have never seen a carcinoma result in a breast from this type of trauma, notwithstanding it is quite severe—more severe many times than is the contusion type of trauma. In a carcinoma of the breast the etiologic condition is different. In the etiologic factors of carcinoma you have carcinoma perhaps more frequently occurring in the breast than in any other position of the body. It can be laid down as a very well-established law that carcinoma is the result of recurrent mild traumas. Let us put it in another way—the result of recurrent mild irritations. The breast is the exception to this. The breast gets its carcinoma from one injury of more than mild severity. That injury is usually severe enough to impress itself on the patient's mind, and for the patient to go on for a year or a year and a half after the injury, when the cancer manifests itself. That is not true of carcinomata in the other portions of the body. Carcinomata in the other portions of the body result, as in the pyloric zone of the stomach, as in the cervical zone of the uterus, from continued or repeated mild irritations. On the lip you have it recurring from repeated mild irritations caused by the use of the pipe. On the knuckle you have it recurring in a crack from repeated mild irritations; but in the breast you have one injury. For instance, the patient is struck by a post or a falling object, or something which attracts her attention sufficiently to impress it indelibly upon her mind immediately, and a year or two, three, or four years thereafter, at exactly the point she received the blow or injury, there are manifestations of malignancy.

PELVIC TUMOR

HISTORY

Female; occupation, housewife; age, forty-five. Married twenty-five years. Admitted December 1, 1911. Entered hospital because of a generalized abdominal swelling. Family history negative, with the exception of mother, who has arteriosclerosis. No malignancy or tuberculosis in the family. Past history, had the ordinary childhood diseases, and diphtheria at the age of twenty-six. Uses tea and coffee moderately. Has had two children—normal deliveries; both children living and well. One miscarriage, after the birth of the first child, which occurred three weeks after the last period missed. No leukorrhea; no history of specific infection. Has always had dysmenorrhea; menses normal in amount, duration, and regularity up to the commencement of the present trouble.

Present Trouble.—Started in August, 1910, at the age of forty-four. The patient stated that at that time her periods, which, with the exception of the dysmenorrhea, had always previously been normal, occurred at irregular and shorter intervals, coming on every two or three weeks, and although the duration of the flow was the same as previously,—five days,—there was an increased amount of blood and many clots. Since May, 1911, the intervals between the flow have increased to five and six weeks, and the duration of the flow decreased from five days to two days. The flow during these two days, however, is profuse enough, so that the patient uses three or four napkins a day. Since September 10, 1911, the patient has been nervous and irritable; has complained of headache and occasional indigestion, and has had three attacks of dyspnea and palpitation of the heart. On November 20th of this year the patient states that her abdomen began to appear larger. She noticed this by the difficulty experienced in getting into her clothes.

This abdominal enlargement has increased up to her entrance into the hospital. She has never had any pain in the abdomen, nausea, or vomiting. Her appetite has been poor. She has been running no temperature.

COMMENTS AND OPERATION

According to the history of this case, the menses began to diminish in quantity; the interval became longer, and finally, in September of this year, her abdomen began to enlarge rather suddenly and assumed the condition of ascites. In connection with this condition of ascites the doctor said that there was no pain; there was nothing to indicate that she had one of the painful conditions that occur in the abdomen which often increase the quantity of fluid therein. The fluid came on without any particular manifestation until the swelling of the abdomen appeared. Of course, this is suspicious of malignancy, and when the fluid is clotted, it is positive evidence of malignancy. We will see, first, whether she has clotted fluid in her abdomen and, second, if it is, what the cause of it is.

I find it is not clotted. This is in her favor. If it were the other way, it would be positively against her. You see a large quantity of fluid. It is not ascites. She has a mass in her right side which is fixed in the pelvis. This mass is nodular. It is not a fibroma of the uterus in its clinical history, and the symptoms are not those of rotation of the pedicle. When there is rotation of the pedicle, the patient has all the symptoms of acute infection in the peritoneal cavity. We will let this fluid flow out rapidly, so as to give us a better field. Here is the mass as we described it. We will put in a retractor to demonstrate it. Here you will see an irregular tumor, fixed on the right side, and that is not the worst pathologic condition you see in this case, for studded over all the peritoneum are malignant nodules. There are papillomatous nodules all along that side. It shows that it is what we thought or believed it was—a malignant lesion.

Now, let us see if it is a papilloma of the epithelial variety, in the cancer class, or whether it is a sarcoma that is of the connective-tissue variety. It is a papilloma grown to the abdominal wall, grown to the pelvis, grown to the sigmoid clear up to the margin of the pelvis, and it is impacted in the pelvis. That means what? That it is an irremovable carcinoma—that it has occurred in the ovary. I feel a small mass on the other side, and I find that the other ovary is in the earlier stage of exactly the same form of lesion. What does that mean? It means cancer of the upper surface. I can show you one of the pronounced nodules. Here is one of the characteristic nodules of a papilloma. You see the typical, warty cancer settled here on the side coming through from the center. We will take it off. Here is one of the epithelial grafts that are studded over all the peritoneum. We will remove this and make a microscopic examination. This case figures out just exactly as we anticipated, namely, that the coming on of that fluid suddenly, without pain, is always an indication that you have a malignant lesion. When it comes on in the other way, with pain, with a stormy attack, it has an entirely different significance.

Let us go into the pathology of this case while we are closing the abdomen. What is the pathology? I will have the history read again, so that I can bring out certain points. [History reread.]

Here was a mass in the pelvis as hard as a fibroma; still you noticed that I did not mention fibroma as one of the likely pathologic conditions in this case. Why? Because there were certain changes in menstruation at forty-five that never occur with a fibroma that is fixed close to the muscularis and which is immovable. That is why I did not mention it. There was no occasion to mention it. It was excluded by the history *per se.*

I will have the history of her menstruation read again, so that I can show you the line which divides the two histories. [Menstrual history reread.] With a fibroma these patients do not go four weeks, but three weeks, sometimes two weeks, and sometimes only ten days, between the flow. In the case of a fibroma one flow might run straight into the other. Therefore you have here a striking difference in the menstruation as compared with a case of fibroma when the patient reaches forty-five years of age. A patient with an intramural or submucous fibroma has her menopause postponed to the fifty-second, fifty-third, fifty-fourth, or fifty-fifth year, but she menstruates with great regularity up to that date. These patients have no time, either six months or a year, in which they cease to menstruate, and in which the flow comes on again. Is that clear? With carcinoma they will have an interval of a year; after they have passed forty-five years of age very commonly there is a year and a half between the two menstruations, and then the flow is greater in quantity than that of normal menstruation, and that is pathognomonic of cancer. (I am speaking now of carcinoma of the uterus.) That never occurs with fibroma. When you come to lesions that involve the adnexa, you have, in connection with the ovary, two types of malignant disease. One is sarcoma—and sarcoma of the ovary is divided into three different classes: First, we speak of the spindle-cell type; second, the small round-cell type; and third, the large round-cell type. The last two are more frequently solid tumors attended with necrosis or breaking down. The former is more commonly a cystic tumor than are the others, and it is the least malignant of the three varieties. It always begins in the connective-tissue stroma or the mesoblastic portion of the ovary, and never in its epithelial cells or in a Graafian follicle. The opposite is the case when you come to the type of carcinoma we have here. Here we have carcinomatous papilloma.

How does this originate? It never originates in the connective-tissue stroma, but always inside the wall of a small cyst, as a papilloma, as a wart, and then undergoes malignant degeneration. The question of recurrence from a malignant wart is not definitely settled. It is certain that in examining for local cysts, even the mother cyst or larger cyst, of the entire number you will find a papilloma that is non-malignant, and close to it you will find a malignant papilloma. So for that reason it is believed that the parent cyst is often a non-malignant papilloma, and undergoes carcinomatous degeneration, which is so common in carcinoma in other positions in the body. They soon penetrate or rupture, and small epithelial grafts of carcinoma are deposited all over the peritoneum. There is no use in taking out this tumor, hazarding the patient's life, when these carcinomata are all over the body.

What I have told you may or may not be true. Why? In these cases occasionally, where a microscopist tells you it is carcinoma, these tumors undergo exactly the changes that take place in warts. You will see a man at one season of the year with a crop of warts all over his body, and without any reason that he knows or that we know of. When you meet him again, three or four months later, he will not have a single wart. Why have they disappeared? We do not know. It is a clinical fact, supported by observations, that they do disappear. Clinically, our observation in connection with this class of cases has been that occasionally one of them gets well and stays well for years after opening the abdomen, as we have in this case. The reason we know not, but the clinical fact is a well-established one. Operation for the removal of these warts that are adherent to the intestine must be done, but you cannot remove all the structure. The tumor is adherent in the pelvis, is large, and the portion of intestine involved is irremovable. Therefore you should not hazard the life of the patient by undertaking such an operation. Will it disappear by exploratory laparotomy? Will it disappear without breaking up the adhesions, or without endeavoring to remove it? It will not disappear. It will continue to grow, fill with fluid, the patient will have to be aspirated, and finally the condition will terminate fatally as a diffuse papillomatous peritonitis.

These are, fortunately, rare tumors. I do not believe I see one of these on an average in two years. With all our laparotomies, this shows they are, fortunately, rare, because they are so malignant—they are very much more malignant than the cystic type of sarcoma.

In this clinic I demonstrated the case of a woman from whom I removed, four and a half years ago, a large sarcomatous cyst which filled all her lower abdomen. About a year and a half ago I removed a second one, as large as, if not larger than, the first, and this last fall I removed a third one of about the same size. She has gone on perfectly happy, and will be so until she gets another tumor, and when she gets another sarcomatous cyst I shall remove it for her. If I can prolong that woman's life, even though the microscopist reports a spindle-cell sarcoma, it is my obligation to do so. You can take off the tumor and free the intestine, which I did on both occasions. The cyst recurred each time in the pelvis, and went through exactly the same type of growth it did originally. The first time it originated in the ovary. The reason I have spoken at such length is because I wanted to accentuate some of the points concerning the case.

REMARKS ON COUNTING SPONGES

The interne, to whom is intrusted the recording of the counting of the sponges, has been on the service only since the first of the month. He has not had many sentences passed on him as yet. Sister Victorine counted the sponges, and she knows the number is right when the abdomen is closed. A report of the number of sponges must go on every history. By that system you are not so likely to leave sponges in the abdomen after the wound has been closed. The only wonder is that sponges are not left in the abdomen more frequently, but by having one party responsible for them, you succeed in avoiding mistakes or accidents to a greater degree than by having a haphazard method of keeping track of them. I make it a business to ask the doctor with reference to the number of sponges removed, because I am teaching all the time and it is very important to do this. If I should neglect to ask him, it is his duty to see that the record shall show the number of sponges. This lessens the liability of accidents.

EXPLORATORY LAPAROTOMY

(Being the Third Operation on this Patient)

HISTORY *

Female; age, twenty-five; occupation, none. Uses tea and coffee moderately. Family history, mother died of carcinoma of stomach. No tuberculosis. Venereal negative. Menstruation began at the age of fourteen; regular, and of the twenty-eight-day type. Duration, four or five days; moderate flow, soiling three or four napkins. For the past year each menstrual period has been becoming shorter, the last one being one day in duration. Date of last menstruation, December 30th. Occasionally passes a few blood-clots. Has had a leukorrhea since the menses began. No pain or burning upon urination. Had the common diseases of childhood. Frequent attacks of tonsillitis, the last being at nineteen. Had an attack of acute articular rheumatism at twenty, lasting two weeks, involving wrist- and finger-joints. Pleurisy at the age of twenty-five.

Present Trouble.—Began one year ago, with loss of appetite, a feeling of weight in the epigastrium, and pain after eating. The pain came on about one hour after eating, lasting one to two hours. Was increased by the character of the food, acids causing most distress, milk and eggs causing less. This pain was relieved by taking food or a glass of milk, relief lasting from twenty to thirty minutes. Vomiting comes on about one hour after eating, when the pain is most severe.

Ejecta is composed of a brownish fluid, but no blood that she ever saw. With the onset of the pain she became tender and distended in the epigastrium, which condition has remained up to the present time. Bowels are markedly constipated, the patient going two or three days without a movement, and requiring resort to frequent cathartics which do not give much relief. The feces are hard and lumpy; have never contained blood, but do contain considerable mucus. In May the pain descended from the epigastrium to the lower part of the abdomen, then, changing suddenly, became colicky in nature, and was relieved by taking food or an enema. The tenderness was also confined to the lower abdomen and epigastrium. During this attack she did not vomit or have pain in her epigastrium. This attack lasted about three weeks. In six months she lost 32 pounds. Since the onset she has become markedly anemic. Has never had night-sweats or a temperature that she knows of.

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* This history was taken January 11, 1911, at the time of the first operation.

Examination.—No tumor is palpable. The point of greatest tenderness is in the epigastric region, just to the left of the median line. Pressure upon the pylorus zone causes the patient some nausea. Vaginal examination shows a complete retroversion of the uterus, causing pressure against the rectum. The cervix is adherent to the anterior wall of the vagina, continuous with it, and is slightly ulcerated. High proctoscope, negative.

Further History, written September 7, 1911

Patient was in hospital in January, 1911, for five weeks, at which time she was operated on. About six weeks after operation patient began vomiting. This would occur about two hours after eating, and was accompanied by pain in umbilical region, griping in character, similar to a pain after taking a physic. Also complains of a sense of distention, which is relieved by eructations. Patient sometimes vomited before breakfast, and at times could go all day and finally vomit at bedtime. Sometimes, too, had pains which would be relieved by eating, which would bring up gas and cause relief. No weight lost; appetite good. Bowels fairly regular, no blood. Never went more than two days without vomiting.

Further History, written December 5, 1911

Patient is a female, twenty-six years of age, who was operated on in January, 1911, for a duodenal ulcer. A posterior gastroenterostomy was performed at that time. She returned on

September 7, 1911, because of continual vomiting, which followed the first operation. On September 9th she was operated on again. She now returns for a third operation, because of pains over the lower iliac region, extending entirely across lower abdomen. The pains are colicky, occur every day, and are only relieved on taking sodium bicarbonate, which assists her in belching the gas. The patient states that she is unable to expel any gas per rectum or to expel any feces without a rectal enema. However, for two weeks following her last operation she succeeded in having normal bowel movements without the aid of enemas. Since that time (three months) she has used an enema every day. The patient states that four times vomiting has followed the pain, which was unusually severe at that time.

COMMENTS AND OPERATION

You have heard the reading of the history of this case. It is very essential to know what was the pathology found at the first operation, in order to clear things up and to see whether or not it has any bearing upon her present condition. There were no adhesions of the viscera. There was duodenal dilatation in the region of the pylorus, which stood out and resembled an hour-glass stomach, which was diminished 50 per cent. in size as compared with the previous condition. The previous ulcer was on the lesser curvature of the stomach and had healed. There was a new one an inch higher up, indurated, and crater-like. If possible, I want to show why she had a return of the symptoms following the second operation. The gastro-enterostomy was patent. There were a few adhesions on the under side of the mass, and it was decided that the symptoms were due to a fresh ulcer. At first she had an ulcer on the lesser curvature, near the pylorus. She had, in connection with that, a thickened duodenum. The original operation was a gastro-enterostomy for the relief of the tension on the duodenum. There was healing of the ulcer near the pylorus, and, notwithstanding the fact that she continued to improve, she had another ulcer, which gave her the acute symptoms for which we operated. This brings out the fact that the last word has not been said in connection with gastric ulcer. A fresh ulcer occurred while she was having drainage and after the original ulcer had healed.

What are the symptoms now? We are told that she complains of much pain after eating. Even taking a glass of milk causes pain about an hour afterward. Her physician informs us that she begins to get relief as soon as she feels gas escaping where the tension was. She did not vomit during the last attack.

In the history of these cases this is the first time that we have had a combination of duodenal and gastric ulcer. The constriction of the pylorus looked more like an hour-glass contraction of the stomach, but the moment you palpate you could tell which was stomach and which was pylorus.

This is an old condition, which was first described by Rokitansky as he found it in the dead-house. The theory advanced by most writers was that the megaduodenum is due to compression of the superior mesenteric artery as it passes across and causes a fixation of the duodenum. That is not our belief of it at all. We believe the condition begins with an ectasis, and with the ectasis comes the kinking of the duodenum. It is not the vessel that produces the compression, but a band of connective tissue.

What are we going to do? That will depend entirely upon what we find. We are going to try to relieve this patient of her symptoms. She has had a recurrence of a pathologic condition that primarily healed.

Notice how cautious I am in opening this, because it is a tertiary opening. We will determine, when we get in there, whether adhesions have resulted under the precautions which we take to avoid adhesions. There are adhesions at this point, although we are not as yet in the free peritoneal cavity. We are simply in a small groove. We will go to the other side and see if we can get at the point there. There are, as you see, not only adhesions *in loco*, but adhesions for a long distance to the side—in every direction apparently.

We have gone through and are now in the free peritoneal cavity. There is the pylorus. As you see it come into the field you notice the results of the dilatation which preceded the primary operation. You see the enormous size of the pylorus as it stands out from the duodenal dilatation that was present there. The duodenum is adherent to the side. This is where the duodenum was freed from the side in the original operation, and adhesion recurred in that position. There is the gall-bladder. Now, we are in a situation to see this greatly enlarged, still dilated duodenum, and when you look at it, it looks like an hour-glass stomach. It is contracted to what it was formerly, and materially thickened. This clears up that situation. Here are some enlarged glands in the neighborhood. See them shining through here, showing infections from the ulcer! Now let us take up this stomach phase. There is no trouble in demonstrating the position of one of these ulcers. There is the position of the ulcer. Let us see how much of that has healed. There is no crater there. There is no induration, but there, beautifully illustrated, is cicatricial tissue where an ulcer has been. Lower down we find a second ulcer, which is active and large and very much indurated. It is fully an inch in thickness, and now I think I can demonstrate it to you. You see a thick bunch standing out. There is a large active ulcer on the lesser curvature of the stomach, involving all the lesser curvature of this organ, and you see the enormous thickness of it. This is not an ulcer to resect if you consider the life of the patient, but if you think of technic, it is fine. Here you see a decided crater. There is an ulcer that has healed on this side. Here is one still lower down. Let us see whether the opening is still patent, and if it is patent, we must keep it so, and not close it up. It will not do the patient any good to close this opening. This patient is having symptoms from the present active ulcer, and it is what we would expect. The opening is amply large from the stomach into the jejunum, and there is no occasion for making it larger—meaning that she has ample intestinal drainage.

Would it heal faster if we were to close the pylorus or if we were to leave it open? In my mind the closing of the pylorus is not a difficult, but rather an easy, thing. There is evidence here of previous ulcer in this neighborhood with a hard firm mass. The pylorus, however, is patent; it is not closed. We could close this and throw all food out of the megaduodenum without a particle of trouble. I am going through the megaduodenum here. We have freed this on the lower side, and now we will free it on the upper side. Now, we are getting through at the line where we are to make the occlusion. You see, I have my line of division here between the duodenum and the pylorus. Here is this large duodenum. We are ready to open the intestine. We get a bleeding from the inverted mucosa. Near the bleeding from the inverted mucosa you can see what you can do. By holding this up in this way you can free it to the side and put a ligature on it, as one would put a ligature on the mucosa of the appendix and then invert. You doubtless remember the splendid work of Dr. Hessert in regard to inversion of the mucosa of the appendix, and we have the same proposition here.

The first thing to do is to care for the mesenteric side. Remember the mesenteric side is the dangerous side in every type of end-to-end union of the intestine. Get the mesenteric side closed, and ligate it before you start your inversion. That is the first part of the procedure. We will tie these vessels that are to be inverted; then we will make the inversion of this the same as of the duodenum. That is to be inverted, as I take no chances on it bleeding inside. It is almost as easily closed as the appendix. This turns in the first row of sutures, and it covers all these raw peritoneal surfaces. That closes the end of the stomach. The end of the duodenum is more easily closed than the end of the stomach. There is, however, a disadvantage in closing the end of the duodenum, in that you have practically no peritoneum covering it. There is the duodenum held up for ligation. You can all see how this is tied. We will tie these vessels here on the side, and then make the same type of inversion that we did before.

We come again to the place of danger. Here is the zone of danger—the part that is not covered by peritoneum. I take up the peritoneum on the under side, catch the muscularis, and make the same overstitch that we have made for so many years in connection with the appendix, so as to cover that as nearly as possible, and make it a circle of peritoneum as near as we can.

Why? First, it ligates the vessel; second, it gives you peritoneum for immediate adhesion, which is the all-important point; third, it protects the cellular space or spaces beneath, which are the dangerous spaces of infection. We will take off the clamps and invert that end which you see in exactly the same manner as one does the appendix. I will take up the next stitch, which is to come over the stump, and embed it. You see, again, that it is embedded with the same simplicity that one embeds the stump of the appendix. We follow this principle practically in operating in every portion of the body. Catgut is used many times for ligatures. This is Pagenstecher linen, which we use for all this type of suture work, because if this should penetrate the mucosa, it would not be digested. Furthermore, if it does penetrate to the mucosa, it has no capillarity and will not carry infection out into the field. The cellular tissue that is the least protected will be covered in additionally, and there are two stumps. We will simply tack these together with catgut on a straight needle, so that these two lines of suture will be additionally protected. You can see that the two lines of suture will be nicely protected by peritoneum in the neighborhood. In doing this we have put the megaduodenum out of consideration. Whether that will hasten repair of the ulcers time alone will show. Whether that will prevent their recurrence time alone must answer. We will not predict.

It is useless in these cases to predict the outcome when we know so little about the etiology of these ulcers. Predicting or guessing is of little value. We admit, and it is admitted, that we know little or nothing about the etiology of these ulcers as yet. They have been attributed to the colon bacillus, but we can say for that that the case is not proved. You see how well these lines of sutures are supported from the side with a double row of Lembert sutures in addition to the ligature. Here, we are sure, we will have organic adhesions because we have organic abrasions inside the peritoneum. We will not have adhesions, but we will have surface adhesion around the gall-bladder where there was adherent omentum. The omentum I am putting over there because this will play a feeble rôle in preventing adhesions. I am making an ectropion again at the point of approximation of the abdominal wall. Fixation by adhesion in the future should play little, if any, rôle. These figure-of-eight sutures I put in before I insert the aponeurotic suture, because here we are unable to outline the aponeurosis clearly. We can outline the aponeurosis now; we pick up the aponeurosis separately, and, as this is a great support to the abdominal wall, at this time we want it accurately sutured.

Under this form of anesthesia, given by Dr. Teter, I have been impressed with the ease and the great relaxation of the abdominal wall during the period of time I was working in the abdomen. It was as nearly perfect as anything could be. There was not the slightest resistance offered by the muscles during all the time I was in the peritoneal cavity. We do not want to give these patients ether at all this morning, but nitrous oxid and oxygen, and so I have to complete the work. This is a horsehair stitch which we use to approximate the skin. The amount of venous oozing is more than the average where an abdominal incision has been made. Whether this is because of the previous operation I cannot say. The first time we operated on this patient a keloid formed at the site of the incision. The last time we operated there were no keloids and no pain. The first time she had much pain, so that she could scarcely touch the skin. Why was that? Will some one explain it? It was in the same individual. We attribute keloids to the individual's peculiarity, but there is something we have not learned about this. We dissected out the keloid in the second operation.

Let the record show that the pylorus was very patent; that the ulcers near the pyloric zone, near the lesser curvature, were completely healed; that there was a large ulcer higher up, and fully an inch in diameter, and nearly that much in thickness; that the gastro-enterostomy was patent; that there were adhesions of the pyloric zone to the anterior abdominal wall, to the gall-bladder, and to the under surface of the liver; that a closure was made of the pylorus by dividing the duodenum just outside the pylorus, cutting the muscularis and muscularis mucosæ, and then ligating the mucosa on the gastric side, and inverting with two rows of Pagenstecher linen, suturing the muscularis and the peritoneal surfaces to the stomach first; the same procedure was resorted to in closing the end of the duodenum. Both of these surfaces were then accurately adjusted so that the space in which we operated was placed entirely subperitoneal, and excluded from the free peritoneal cavity. The abdomen was closed in the usual way. The anesthesia was conducted by Dr. Charles K. Teter, of Cleveland, Ohio. It was nitrous oxid gas and oxygen anesthesia, otherwise the patient would not have come out from under its influence so rapidly as she has at this time.

FRACTURE OF THE PATELLA

HISTORY

MALE; occupation, laborer; age, thirty-nine. Admitted November 26, 1911.

History of Accident.—Accident occurred November 26, 1911, while unloading heavy castings from a box-car with a two-wheeled truck. He attempted to force the bottom of truck under a casting which jarred the truck back against him, throwing him to his right knee upon the floor. At the same time he attempted to catch himself by forcibly extending the left leg, and was struck on the left patella by the truck while the extensors of the leg were tense. After being struck on the left knee, he fell to the floor and could not rise or extend his left leg.

COMMENTS AND OPERATION

From the history you will see how long we have waited in this case. The only danger in these cases is in operating too early. After he came in we injected 20 c.c. of formalin and glycerin into the joint, and he got an inflammatory reaction in the capsule or synovial membrane of the joint. What does it do? It infiltrates the synovial membrane. It stimulates the production of the polymorphonuclear cells within the capsule; it coffer-dams all the lymph-spaces.

Now, then, we are going into what? Not a fresh wound, in which all the tissues have a *minimum* resistance against infection, but a granulation wound, where all the tissues are coffer-dammed to *resist* infection. That is the beauty of doing everything in surgery. What would I do if that were on the surface? What would be the conditions if that were on the surface, looking at it in a rational way? I am able to rub the ends together, showing that we have no interposition of material, or at least of the capsule, the condition that prevents union.

I am now going in below. There is the lacerated capsule; there is the blood-clot or the hematoma. There is the clot within the joint, which I am not going to touch. I do not wish to do what? To disturb that synovial membrane. I want to show you the condition here. There is the freshened surface of the bone of the lower fragment. Let us see where the upper fragment is and where the capsule is. This is a beautiful thing to see. I want to show you the capsule—how it is rotated over the upper end and how little. There is a portion of the capsule, and you see it has covered fully two-thirds of the articular surface of that patella. That is why we do not get union much more frequently than we do, and that is the cause of failure of union much more frequently than is the fact that these ends are so far apart.

I am not going near the synovial membrane at all. I am getting under this turned-down portion of the patella. There has been bone softening; there is a granulation tissue mass. I have the greatest possible respect for that joint. Here is where we are covering the end—to prevent what? To protect our bony surface. There is a stellate fracture there. I am getting into the quadriceps tendon above. You see that is not going into the bone at all. I am raising a button of bone for what purpose? To prevent my wire from cutting, that is all, putting it right through the tendon and using the button of bone to prevent it from cutting. Here is the fibrous capsule on the side of the aponeurosis of the patella. It cannot become again interposed. There is the lacerated extension of the vastus internus, as you see, united as it goes down into the aponeurosis over the joint and on both sides of the patella. This is the vastus internus and, on the other side, we have the vastus externus.

Let the records show that there is a stellate fracture of the patella, dividing it through into almost two equal portions. You can see that we had practically nothing at all in that joint—only one sponge touched it. The reason for this you all know. In our experimental work we showed that so long as you kept the endothelial layer of the synovial membrane intact, you had little or no danger of infection. You know that in our experiments on dogs we found that if we would inject a syringebarrelful of infected virulent pneumococci into the knee of a dog with a hypodermic needle the dog would have no trouble from that injection unless you traumatized the joint. On the other hand, we found that if we injected the other half of the same syringe-barrel into the other knee, and at that time just irritated the synovial membrane and cartilage to a degree that one can do it with the ordinary hypodermic needle, a fatal phlegmon would set in, and the animal went on and died. It took a quarter of a century to find out this principle with reference to the peritoneum.

Here is another serous surface that must have its endothelial cells respected just as much as the serous surface of the peritoneum. This was the type of operation that Lord Lister first did in London, England. He came down from Edinburgh with his ideas of antisepsis, and this was one of the first operations he did in London to demonstrate his confidence in the method which he was advancing, and to a community of practitioners of medicine who were most incredulous. Lister did just this type of operation with perfect asepsis, and did it with results which he frequently secured thereafter. Every man who witnessed the operation shook his head and felt sure the result would be unfavorable, but Lister had no misgivings. He demonstrated conclusively and unmistakably what he could do and sustained his position. I will say this of the London profession, that when they are convinced, they are fully convinced, and what Lister did shows not only the courage of his conviction, but he displayed the courage of experience which he was justified in showing. I do not know how early Lister demonstrated that the work of opening joints with the antiseptic method was better than the aseptic. Just why I do not know.

We use carbolized gauze in this case. In this case we have more or less serous discharge,—bloody discharge,—and we must provide for the care of that to render it sterile after its escape, otherwise you have trouble from it where it goes onto the surface of the skin, differing from peritoneum. Why? When you close the peritoneum it is closed, and closed for good. There is no danger of oozing or anything else. Here there is always some oozing, but there will be very little synovial fluid forming in this joint. Operating at this time you will not have hydrops, but might have had if you had operated immediately. By operating on these cases immediately you would expect considerable effusion into the joint, and that would interfere with the process of repair by separating the tissues.

We will not put on a dressing. Buck's extension will be applied, and allowed to remain for about four weeks; then the inflammatory condition will practically be all over.

BLOOD-CLOT IN THE BLADDER

(TUBERCULOSIS OF RIGHT KIDNEY)

HISTORY

MALE; occupation, waiter; age, nineteen. Admitted December 3, 1911, complaining of pain over right upper quadrant of abdomen and frequent urination. Urine contained much blood. Family history: Mother had an operation in Italy. Right kidney was removed. Made good recovery. Patient does not know the cause of the operation. Has a cousin in the hospital at the present time with kidney trouble. Past history: The ordinary diseases of childhood, otherwise has always been well. Uses coffee, but no alcohol or tobacco. Is unmarried. Absolutely denies venereal infection.

Present Trouble.—May 28, 1911, the patient had a chill during the night while in bed. The chill lasted two hours. Did not feel feverish, but experienced a slight pain over the right upper iliac region. He had to urinate 20 to 30 times during the day and 4 to 6 times during the night. He states that the color of the urine was normal at that time. He continued at his work at this time, and states that he had no sore throat, cough, nausea, or vomiting at this time. He continued to have this frequent urination for three months,—up to September 1st,—and then for the period of one month had no frequency of urination. On October 2d the patient states that he had another chill during the night while in bed. He had moderate pain over the right upper quadrant of the abdomen. The abdomen was tender. The chill occurred first, then the pain immediately occurred, and was followed by vomiting within ten minutes from the time of onset of the pain. He did not feel feverish at this time, and was not confined to his bed. The pain over the right upper quadrant, which is dull and aching in character, has continued up to the present time. Patient has been easily fatigued, and has lost 12 pounds in the last six months. On December 2d the pain became a little more severe than usual, and the patient was forced to rise at 4 A. M. to urinate—the urine was bloody. The patient was forced to urinate four times between 4 A. M. and 7 A. M. He states further that he urinated 10 times during that day—December 3d. This pain, frequent urination, and blood in the urine have continued up to the present date. Leukocytes, 13,000; Urine, field packed with red cells. Remittent temperature—never over 101° F.

COMMENTS AND OPERATION

This patient has an enlarged right kidney. It is sensitive. He gave a positive response to tuberculin. Cystoscopic examination disclosed a large mass in his bladder, brownish black in color, and the question arises, Is this a neoplasm or a clot? The cystoscopist believes that it is a neoplasm. To my unaided vision in that line I would say that I could not tell whether it was a clot or a neoplasm. It looked black enough to be a clot. As I have said, he responds to tuberculin positively. He has an enlarged kidney on the right side. Is the kidney enlarged from tuberculosis, is it a cystic condition, or is it a papilloma growing over his right ureter? These things are to be settled by this operation, which will be in every sense an exploratory one. If it is a neoplasm, then it is doubtless of the papillomatous type, is non-malignant, and should be removed. He has been bleeding profusely. There have been large quantities of blood in his urine every day.

There, you see, is the symphysis we are dividing through here. The recti are on tension. There you have the heads of the muscles on each side. We will divide the head here close to the bone, and allow it to retract and go into the space of Retzius. There is fatty tissue in the space of Retzius. The head recedes, and we have ample space for the opening of the flap. We push the peritoneum up. In doing this I always get clear under the symphysis, or well under the symphysis, making the opening close to the bone. Here I have the bladder fully under my view. Here you see the muscularis shining through the field. The next thing is to get hold of the bladder and hold it steady, and for that reason we put in a stitch going through the wall of the bladder, and that has on it a double thread. Here is the second stitch, put in in the same way.

We open up the space of Retzius, and we have a large cavity, which is entirely unnecessary. There is the bladder anchored above the stitch; we steady it on both sides, and divide that on the symphysis side, to be sure that I am not going into the peritoneum—that I am going into the bladder. There is bloody urine flowing out of the bladder. In doing this operation you are in no more danger than in making an opening as large as that in the thigh. Every move is mathematical. These are the two bladder retractors we use, and they are splendid for this purpose. We will get all this urine out. There is a blood-clot here, and those who are near enough can see this large blood-clot at the bottom of the bladder. It is a large black blood-clot. There is a part of it, and there is the other part.

Let the records show that our tuberculin diagnosis was correct: that there is no tumor in the bladder. There is a small ulceration in the bladder at the fundus or just back of the bas-fond. It is quite a large ulcer, and that which the cystoscopist thought was a tumor has proved to be a clot which will have to be removed. I have now removed this clot, and we shall close with catgut. We cauterize the base of the ulcer, and commence closing the bladder-wall while they are getting the cautery ready. This catgut does not go through the mucosa, but muscularis. The ulcer is not in the trigone, but it is back of it, on the wall of the bladder, and the remaining portion of the bladder-wall is in splendid condition. We will now apply the cautery. This will stop the bleeding from that point. I am now taking out the anchoring suture and approximating the muscular layer with catgut. We will put a drain in the space of Retzius and not into the bladder.

We will return again to our original diagnosis of his right kidney, with secondary tuberculosis in his bladder and a tuberculous ulcer in his bladder. We will not take out the kidney now, but later possibly, when we have evolved a resistance from giving tuberculin. If he responds to the tuberculin test, we will take out the kidney. Let the records show that there is no particular congestion or irritation around his urethra. This gutta-percha tissue I think will act as a drain in the space of Retzius, and then he will have a much better union and necessarily a better result than he would have if I were to put a tube in his bladder for the purpose of drainage. A tube in the bladder is a source of irritation, and in many instances leads to trouble. I suture the gutta-percha tissue in.

The history of this case fits in with the pathologic condition, and what the cystoscope showed was not a papilloma, but a clot of blood. [History of the case reread by the interne.]

The history is fairly good, with the exception that it does not state anything more definite about the chills. The patient had malaise and exhaustion. He was a little more flushed, and had a little more fever in the daytime for something like three months, and went along in this way until he had a chill. The one thing that does not fit a hemorrhage from the tuberculous kidney is that the primary chill is without hemorrhage the next day. He should have had a hemorrhage after the chill. A chill is evinced with tuberculous ulcer, with a mixed infection approximating a large vessel, which he finally had, and it opened with a pressure hemorrhage which he has been having. Chill and high fever are common in conditions in other portions of the body preceding a hemorrhage, whether it be a tuberculous ulcer of the intestine, a tuberculous ulcer of the urinary bladder or of the renal pelvis, or whether it be a tuberculosis of the lungs. The patient has a chill, not so severe in the lung, nor more severe when he has tuberculosis of the intestine than tuberculosis of the kidney; it indicates that infective material is working on the wall of a vessel. When a patient with tuberculous ulcer comes to you and tells you that he has had a chill, then commence inspecting his feces for blood. When a patient tells you that he feels cold, and finally has a temperature ranging from three and a half to four degrees higher than normal, look out for hemorrhage in the next forty-eight hours. Tell him to lie down. The ulcer is eating its way through the wall of the vessel, and septic matter is being introduced into the blood-current.

This man's hemorrhage was slow. It should have come with the stormy manifestations, because there is not a particle of doubt in my mind as to the association of the pathology with the clinical history. That is where it belongs. His temperature was 103.5° F. preceding this bleeding. The next day he walked about with his urine full of blood, and the blood-clots had been there since.

CHARCOT'S DISEASE OF THE HIP JOINT

HISTORY

Male; occupation, electrician; age, forty-one; married eleven years. Admitted November 4, 1911. Family history: Uncle died of pulmonary tuberculosis. Aunt died of carcinoma of breast. Otherwise negative. Past history: Ordinary diseases of childhood. No typhoid, scarlet fever, diphtheria, or pneumonia. No jaundice or tonsillitis. Shooting pain in legs quite frequently, especially at night. No previous operation or severe injuries. Patient smokes 6 to 8 cigars a day, but uses no alcohol. Neisserian infection occurred three years ago; duration, six months. No remaining symptoms. Denies any primary lesion; no history of secondary symptoms.

Present Trouble.—Dates back about six weeks. Patient states that about six weeks ago,—he does not remember the exact date,—while wheeling a wheelbarrow full of dirt over a plank, his left leg slipped off the plank and he sprained his left ankle. This was not severe; he continued his work for one and one-half hours after this, and rode his bicycle home—a distance of three-quarters of a mile. He continued his work for two weeks. During this time he limped on his left leg. He had no pain and swelling, and could attend to his work. The only thing he complained of was that he could not handle his left leg as well as formerly. Two weeks after injury, one morning, on getting out of bed, he noticed that his left hip and leg were swollen. Says within twenty-four hours his left hip and entire leg were swollen to twice their natural size; that he had no chill or fever; no pain in leg. Could press on it as hard as he wished without pain. Could move it without pain. He remained at his home for two weeks, and leg gradually reduced in size. Says he was

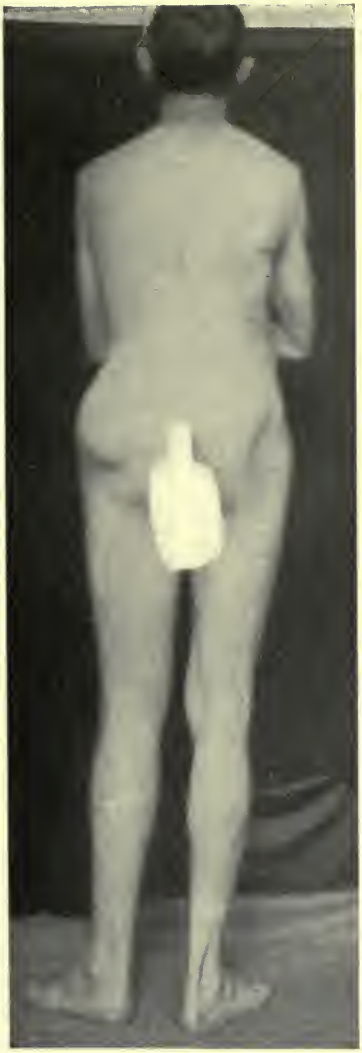

Fig. 4.—Charcot's disease of hip-joint. Back view, showing enlargement.

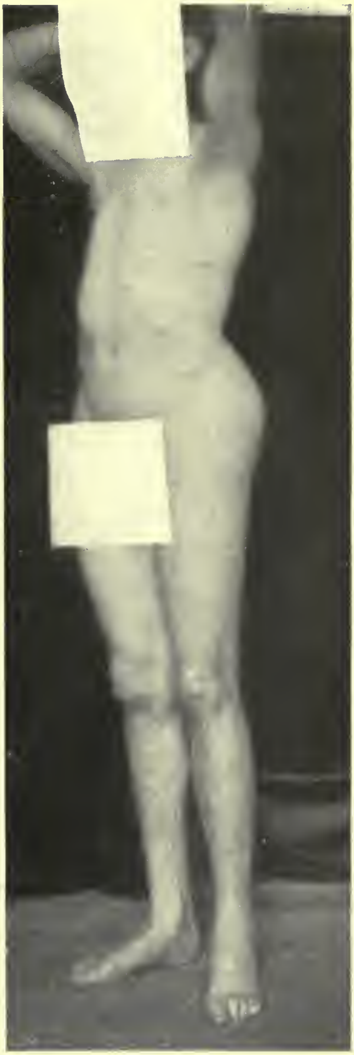

Fig. 5.—Charcot's disease of hip-joint. Side view, showing enlargement.

in bed part of the time, and up a considerable portion of each day. Says leg was swollen at night, and in the morning swelling was almost entirely gone. During the remaining two weeks, up to the present time, he has been about. Says hip has remained swollen, but has never been tender or painful when he moved. He says it troubles him when he walks, in that he cannot handle it as good as formerly—that he often stumbles, and that his foot seems to be slightly thrown out from date of injury, six weeks ago. For past four weeks he has been walking on crutches, not because of the pain, but because of uncertainty of position of his left leg, of which he does not have perfect control. Says on second week after his injury he noticed a grating sensation in his left hip—no pain whatever. Says he has all motions of foot and leg.

General health and appetite have been good; bowels regular. For past three months has lost about 10 pounds in weight. No night-sweats. His left leg is shorter than his right leg.

Examination.—Large mass of bone near the joint appears to be a detached independent mass of bone, separate from femur. Thickening on inner side of ilium. Roughening and thickening near trochanter. Measurement 2 cm. short.

COMMENTS AND OPERATION

As a result of an injury to the hip this man was incapacitated for working for two weeks, and finally he quit working, not because of pain, but because he was uncertain as to the position of his limb. He came to us for examination. When you look at this hip I want you to notice the enormous size of the tumor. It is smaller now than it was, but all this came on in six weeks. There was but one conclusion to come to concerning a growth of this kind from the statement of the doctors and from physical examination: this was that it was a sarcoma of the hip, and that was the conclusion we arrived at after making a careful analysis of his case. He was about to depart from the office when I asked him again why he limped. He replied, "I limp because I do not know just where my foot is going." I asked him whether he had pain, and he said, "*No; I have never had a particle of pain in that hip.*" The x-ray showed an enormous reproduction of bone in the neighborhood, but did not show the line of fracture. It showed that the trochanter had gone up as though the neck had melted down with the neoplasm, but he did not have a particle of pain in that limb from the beginning. He fell over from his bicycle and struck on his trochanter.

Two weeks before this man came in we had operated on a case of sarcoma of the femur which resulted from an injury similar to this. This is a nasty case for operation. You will see a large osteoma or osteal production. There is an enormous mass of bone produced outside of the joint. Here is the head of the femur an inch below. When he said he did not have pain at any time it dawned on me, for the first time, as to what was the matter with this man. It was not a sarcoma, but it was a Charcot diseased bone, with fracture of the neck, and that this enormous mass which we feel here was an osteal production the result of his spirochetic infection. This is all bone, but when one gets at the joint I do not know myself. It is going to be a difficult task to get to the joint with the enormous amount of bone deposited here in the histologic structures—those capable of being converted into bone, namely, the wide fibrous tissue that occurs in the mesoblastic membrane.

That is capable, under certain conditions, of reproducing bone, and here is this great mass of bone above the trochanter developed in the bursa of his trochanter. It is a bursal development of bone and a capsular development of bone that does not occur except in pathologic conditions. It resembles the production of bone, and is like that which occurs in the formation of the face. Remember, in the formation of the face you have the deeper layers of the endoderm carrying with it the osteogenetic fibrous layer that reproduces or produces the bones of the face. You have exactly the same structure in the lining of the bursa and in the secondary portion of the fibrous capsule. These are capable of undergoing osseous changes in pathologic conditions from certain types of irritation.

I am going to have some trouble in getting at this joint. I

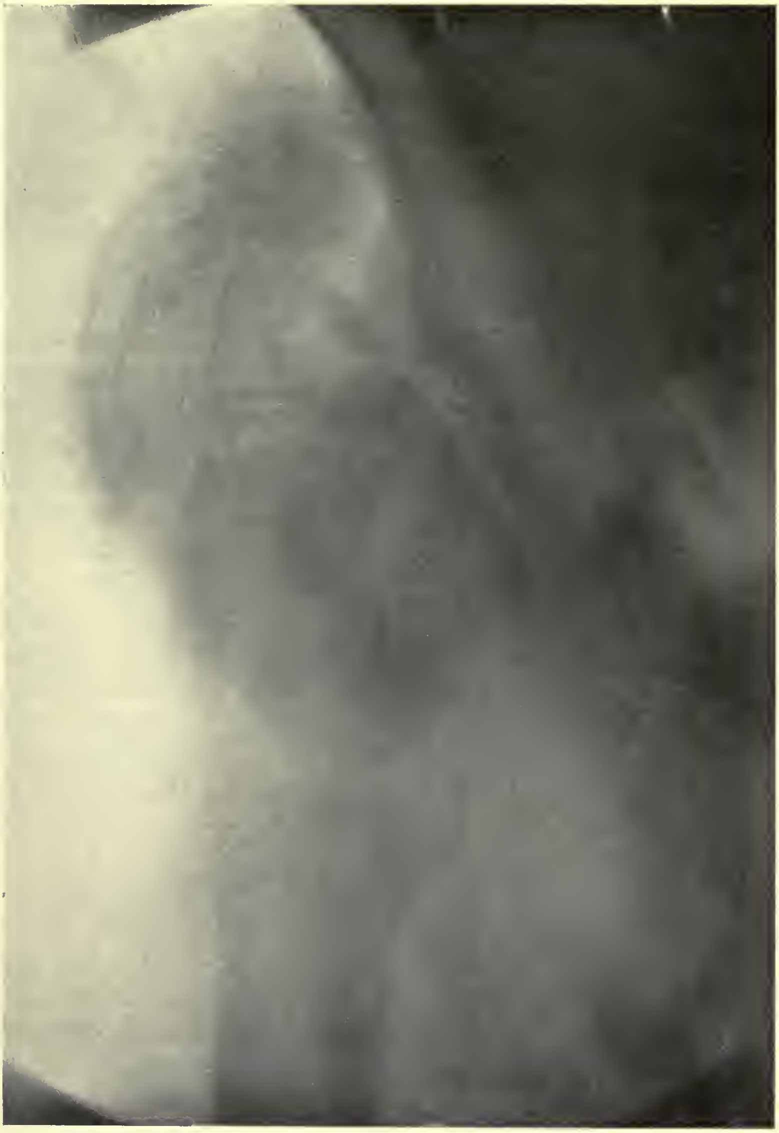

Fig. 6.—*x*-Ray showing Charcot's disease of hip-joint.

am going at it just as though I was going to do an arthroplasty, which is my way of attacking these joints. Here is the anterior superior iliac spine at this point [indicating], and there is the highest point of his trochanter. This is so extensive a growth that a little bleeding from many points will amount to considerable before the operation is over. If we can get the bones in apposition again they will unite. That is the rule that governs our action in these cases. If we cannot get them in apposition, the case will run the course of a non-approximated fracture of the femur. Here we are on to the true neoplasm, and it is in the deeper muscular layer. There is the trochanteric bursa, edematous, and has a deposit and infiltration in it. This muscle, as you see, is a part of the bone. You see it is in the loose infiltrated condition that you observe in sarcoma. It is as firm and as hard as normal bone. There is that loose plaque of bone that is shown in the skiagram. It does not look in the least like a sarcoma. Look at it! It is hard formed bone and in a great large plaque. I had to put that out of position in order to get to the joint. I can see now that the physical findings resemble the clinical history. There is an effusion from the joint. I will now deal with the trochanter so as to get accurately into the neck. Here is the trochanter, and there are the muscles attached to it, and now, while I continue working, I will turn the talking over to Dr. Teter.

REMARKS INTRODUCING DR. C. K. TETER, OF CLEVELAND

Dr. Teter has developed a very well-deserved reputation for his results in anesthesia. You have witnessed a demonstration of his method of anesthetizing patients this morning. He will describe the method to you, that you may have a better appreciation of all its details. He very generously consented to come here today, all the way from Cleveland, to show us what he is doing in this particular line. I think we can truthfully say that we have not, as a profession, paid sufficient attention to anesthetics; that we have not given the subject of anesthesia the serious consideration it deserves, and, as a result, we have had some very unpleasant things to record. I can say for American surgery and for American surgeons that I believe up to the present time the best anesthesias have been conducted in this country, and that we have had a lesser mortality and have had better anesthetic results. Dr. Teter has added another chapter to the advancement made by American physicians and surgeons in this particular line, and it is with great pleasure that I present him to you this morning. Gentlemen, I present to you Dr. Teter, of Cleveland. [Applause.]

REMARKS OF DR. TETER

I am at a little disadvantage this morning in that I have contracted a bad cold, and I will not tire you by talking too long. I hope, however, you will be able to hear what I have to say.

With this anesthetic there is nothing new, so far as the nitrous oxid and oxygen is concerned. It was right here in Chicago that this combination was first given in 1868 by Dr. Andrews. But there has been considerable that is new in the technic that has been developed in the proper administration of this anesthetic. I think the reason that it has taken so many years for this anesthetic to reach a place in general surgery is due to the lightness of the anesthetic properties of nitrous oxid. You know that 3 or 4 per cent. chloroform is strong enough to bring about the desired results from the standpoint of anesthesia. Ether is about seven times the strength of chloroform, while nitrous oxid is given in from 85 to 95 per cent. pure in order to bring about that degree of saturation which is necessary to produce surgical anesthesia. That being the fact, asphyxiation enters into this procedure, and this has alarmed surgeons and physicians to such an extent that it has not been developed for all these years. I noticed an article the other day by Dr. Bevan, of this city, in which he says that it is not a good anesthetic, or that nitrous oxid is not a good anesthetic, in any heart lesion. I thoroughly agree with him when he says that with regard to nitrous oxid anesthesia. If he means nitrous oxid given with air in order to produce this asphyxiation then it is not a good anesthetic, because of the great amount of venous engorgement you have in these conditions. But with nitrous oxid and oxygen it is a different proposition entirely.

Oxygen is the whole thing in making nitrous oxid the practical anesthetic in general surgery. It is easy to explain to you why when I tell you that we have to give from 85 to 95 per cent. pure nitrous oxid. If you are using atmospheric air, if you have a patient you give him 95 per cent. nitrous oxid in order to get him thoroughly relaxed; if this gives only ten parts of atmospheric air, only about one-fifth of that being oxygen, you do not get much oxygen necessarily, and the normal process of oxidation is greatly interfered with. If you use oxygen and nitrous oxid, you have 90 parts of nitrous oxid and 10 parts of *pure* oxygen, which eliminates the asphyxial elements that you depend upon the air to overcome. You can easily see the advantage of giving pure oxygen instead of trying to give atmospheric air.

I will not go into the experimental work that has been done in connection with nitrous oxid, because you have men here in Chicago, such as Dr. Graham and others, who have done a great deal of that work, and you are probably familiar with it. It is a well-known and established fact that from the standpoint of the patient it is the safest and best anesthetic to give, because it does not interfere with the normal elements of the body to any appreciable extent. The things I have developed, not alone with my own experience, but with that of others, in bringing about results are practically four. The first and most important procedure in bringing about surgical anesthesia and maintaining an even degree of narcosis is the ability to give nitrous oxid and oxygen in a perfectly even constant flow.

The most important part of our procedure is the ability of getting the gases to flow perfectly constant, and as often as may be indicated by the symptoms displayed by each individual patient. After you find out the proper amount of nitrous oxid necessary to produce the desired depth of narcosis for each individual case, it need not be changed practically throughout the whole operation, whether it be fifteen minutes or three hours.

The degree of anesthesia and narcosis is controlled by the amount of oxygen you allow to flow in. You keep a delicate stream of oxygen flowing all the time. Practically, it is 10 parts of oxygen to 90 parts of nitrous oxid, varying slightly one way or the other. If the patient is too profoundly under the influence of the anesthetic, you increase the flow of the oxygen and you will see a lower degree of anesthesia, and if too light, you decrease the flow of oxygen and the patient will soon pass into the deeper stage of anesthesia. The ability of adjusting this flow accurately will depend upon the success of the administrator. The less you change these flows the better it is, all things considered. It is far more important, in order to get an even narcosis, that you give an even flow of nitrous oxid and oxygen than to keep adding on either, because the effect of the anesthetic is very evanescent; these patients are in and out in a few seconds. It is a well-known fact that the blood at normal temperature will absorb nitrous oxid about 40 volumes to 100 volumes of blood, but by increasing the temperature of the anesthetic you increase the absorbability of the anesthetic. I am talking now about nitrous oxid. I have not carried out experiments very much with ether in that regard, but I think what I say with reference to nitrous oxid will hold good with ether, regardless of the article that appeared not long ago by a friend in St. Louis.

We have a method of raising the temperature of this gas from 85° to 90° F. I have carried out a great many experiments in my work with a view to getting the best temperature to administer the anesthetic, and I went on the supposition that if I could give the anesthetic at about the temperature of the blood, that would be the best temperature of the gas for administration. But I have found in those cases that my patients came off the operating-table more exhausted than those cases where I did not give the anesthetic at that high degree of heat. Also that the temperature was raised from one to three points, so that I dropped that and am giving the anesthetic vapor from at 85° to 90° F. That does not cause any exhaustion of the patient, and does not raise the temperature of the patient. That is the second point.

The next point of importance, I think, in bringing about surgical anesthesia with nitrous oxid and oxygen is the ability to increase the intrathoracic pressure by the anesthetic. Just how nitrous oxid is taken up by the blood is not definitely known, but it is generally conceded to be by the simple process of absorption. There is no chemical combination whatever. This being the case, it carries out a theory that, by increasing the intrapulmonary pressure, you increase the pressure of the gas, and by increasing the pressure and leaving the gas flow you can deepen the narcosis, and by decreasing the pressure you lighten the narcosis.

A fourth procedure we use in bringing about surgical anesthesia with nitrous oxid is a certain amount of rebreathing of the gases. Some claim benefit from the increased amount of carbon dioxid which, as Professor Hanson claims, is a normal stimulant of the respiratory center, but with nitrous oxid and oxygen I find that there is a slightly increased amount of carbon dioxid in the blood anyway, so that the principal reason why I have used rebreathing for six years is that I have been able to save a great amount of my gas. If we do not allow any rebreathing of nitrous oxid, we have to furnish the patient the entire amount of air he is breathing. We have furnished about 300 gallons of nitrous oxid to 40 or 50 gallons of oxygen, but by using rebreathing we use, in from an hour to an hour and twenty minutes, 100 gallons of nitrous oxid and from 20 to 25 gallons of oxygen. That brings the cost down very materially. It would be almost prohibitive, from the standpoint of price, if you put nitrous oxid and oxygen in small cylinders and if you eliminated rebreathing. It makes the cost from six to seven dollars an hour, while in the way I am giving it it brings the cost down to one dollar or one dollar and fifty cents an hour.

I think that is all I have to say on this subject, and I thank you very much for your attention.

CONTINUATION OF COMMENTS BY DR. MURPHY

The head is practically entirely absorbed, and I show you how much was left. It is split right across the middle of the bone, leaving the ligamentum teres almost intact. Here is a fibrous mass which I will cut off with the scissors and show you what is left of the ligamentum teres. This man had a syphilitic chill, which gave him a triple X Wassermann. The pathologist said he was in the secondary eruption of syphilis. He was, but it was a *bony* eruption. That shows the value of the test as an indicator of the activity of the disease. It was reproducing all this bone. It has left a bunch of bone on his ligamentum teres which I must take out. The head is clear out of the capsule. There is the joint cavity, and there is what is left of the bone—just a little fibrous mass in the ligamentum teres. The head was put in here [indicating] and has passed out of the cavity. I am going to make an escape for it; I will run it back into the capsule. I do not wish to leave this piece of bone or this fibrous tissue, which might reproduce bone, because then the patient could get an ankylosis—a thing which would not be desirable at all. I believe we can get a movable and serviceable joint in this case. There, you see, is the stump of what is left of the ligamentum teres and the fibrous tissue connected with it. We have one cartilage. I have provided for an arthroplasty in starting this operation, not knowing myself what I would do. There is the acetabulum—the bone coming out in a pocket. I am going to put that right back into its capsule.

There is the new rounded head that looks about as normal as the head of a femur can be. That is made automatically with the new instrument we have for modeling the head. It is about as near a normal head as it can be, and it works out very well with the little machine we use. Originally we did this with a chisel and we thought we did fairly well. I make a skin for this head, so that when the doctor [assistant] rotates it, I have it back into the cavity. Now I will suture it in the cavity and retain it there. I will close up the joint around this neck. There, you see, is the capsule. This is a typical result of spirochete infection, and one of the classic things about the early stage of the clinical history is that he had a fracture of the neck of the femur, and yet went right on with his work, which is characteristic of the Charcot condition.

I am shortening up the capsule by an overlapping flap, so as to give him as much support as possible. We secure the capsule very firmly around the head, so that it cannot become luxated and pass up into the side of the femur. Now we have the capsule wired, and we will nail the trochanter back into position. All this suture material is of the phosphor-bronze type. That puts the trochanter muscles all in position. We will suture the aponeurosis here to the vastus externus with an overlapping flap that will give it additional strength. If the head had not been of such shape as it was here, we would resort to a plan which we followed in another case of fracture, namely, that when we got inside we found the head had been absorbed, and not only that, but the neck was clear gone to the neighborhood of the trochanter. There we had to make a new head. There had been a fracture out near the base of the neck, and we had to make a new head. We used to make this new head the anterior portion of the trochanteric zone with its fibrous capsule. We chiseled that off, turned it around, and nailed it to the head, driving the nail from the joint side in, making a new head and implanting it, and it has all healed in. This was done five or six weeks ago. There have been no unpleasant symptoms. The *x*-ray shows it is in there and nicely arranged in there.

We have a new wire mixture of copper and bronze that we are using today for the first time in a regular way, and it seems to us, with a change that we suggested in it recently, that it will fulfil the indications for a ligature. We have to put in some ligatures, and we will put on this finer wire; then we will be ready to bring the flap back and close it up. There is no part of the technic of this which is difficult when one adopts the plan of elevating the trochanter. You will see how soft and thread like this wire is. This is on account of the quantity of copper in it. It is very fine. The danger of breaking the other type of wire is not in the first knot, but in the second and third knots. If three knots are unnecessary, you can pull the first knot fairly well, but when you kink it in the second knot, that is where the danger comes. In the technic of this operation we had to remove what was left of the head, and we had to prepare a new head, that is, a new rounded surface for articulation. There is no effort at regeneration of bone on the end of that new head. There is no effort at regeneration of bone to produce union there, so I have no fear of ankylosis in that joint. It should not occur and it will not occur. Much of the bone which is deposited around at the sides will be absorbed.

This man came to me with a diagnosis of sarcoma, and I concurred in it until he mentioned one manifestation, namely, that he had received this injury *without pain.* Remember, the skiagram was of no value at all. It did not show a fracture. It misled us, and the one fact that he had *no pain* at any time was the thing that enabled us to make a correct diagnosis.

A few days after this man came in another man came in with a typical Charcot ankle. He has left the hospital now. He has received salvarsan, and we put him on bichlorid of mercury injections following the use of salvarsan, and we hope in that way to cure him of his specific disease, and then operate on the ankle. He had had a Pott's fracture and had been discharged from the hospital he was in cured and with a splendid result. He told the operator that a good result had been secured. Immediately following that, after he began to walk, the ankle began to turn and to become more and more deformed. He then criticized his doctor and received some liberal support in this direction, I am sorry to say, from members of the profession, and unjustly, too, because the doctor was not guilty and should have been protected instead of criticized. You remember the etiology of a Pott's fracture and what occurs in connection with it, and you will find that there is an eversion of the foot, and if the case is badly treated, it will result finally in an eversion of the foot; but this man came in with an inversion of his foot and an eversion of the lower end of his tibia, meaning he had—what? That he did not have a bad result from his Pott's fracture; that the condition which developed was *coincident* with his Pott's fracture. He had all the symptoms of tabes except the ataxia of gait. Everything else was exactly right. He had the Argyll-Robertson pupil; he had the absence of his patellar tendon reflexes; he had changes in his sensory zone in the extremities, and everything to corroborate the diagnosis except the ataxia of gait, and such a diagnosis had been made by the doctor, who felt he had had a bad result. The *x*-ray showed a typical Charcot ankle, just the same type of deformity, a sliding by and absorption of the lower end of the tibia, with a marked luxation, with an eversion of his ankle and an inversion of his foot; meaning that it was not a sequel of the injury, but a coincidence of the injury influenced by the spirochetic infection. Also he had one symptom, namely, that he had never had at any time any pain in his ankle. From the moment it was broken he had no pain. He walked on it immediately at the end of seven weeks, which is the proper time.

Let the record show that the upper surface of the neck portion of the head was completely covered with cartilage, smooth, and glistening; that it had flattened out somewhat and was enlarged at its margin; that there was no effort whatever at the production of bone from the surface; that there was some such effort from the periosteum around the neck. This crescent of bone was cut off with a chisel; the head was modeled to the new shape with our head-modeling instrument. In this connection I will say that we have a similar instrument for modeling the acetabulum in cases of ankylosis where we chisel out a new head. The limb will be dressed in an abducted position and put in a rain-trough splint. We will put a large dressing on, approximate the dressing closely, and endeavor not to open it up for ten days or two weeks.

COMMENTS MADE WHILE SHOWING CASES PREVIOUSLY OPERATED ON

1. We took out the stitches in a case of ankylosis operated on by us three weeks ago today. There was quite a little hematoma, and we extracted the fluid through a needle. But we had complete primary union.

2. Here is a case where we did the original operation on the seventeenth of June on the other hip. We have made two movable hip-joints. He had developed ankylosis of both hips, and they were ankylosed in the position I show you, so that he could not walk. He was stiff from his cervical vertebræ down

to his knee. I will show you the result of the first operation, and I will show you now the early result of the second operation. Here is the result of the operation we did three weeks ago today. There is complete primary union. I want to show you the mobility of the hip which has been obtained in three weeks.

3. Here is another patient who was operated on in June. The joint is perfectly mobile. This was a case of bony ankylosis on both sides. This man was operated upon three weeks ago this Monday. He had bony ankylosis of the elbow from infection. He had a compound fracture; he had cellulitis that involved fixation of his joint below, and there you see is the bony ankylosis which we did not touch. He is able to move his fingers somewhat. Motion will improve every week, and we will have to do some operation for the wrist. He will get practically 80 per cent. of motion. His result is now absolutely assured. He has had a second operation, so that you know what the final result in that case is going to be. He is going to have two flexible hip joints. He will walk and sit. He could not sit before. He was perfectly straight.

The trouble in this case was due to a rheumatic infection. I do not believe there is any such thing as rheumatism. Let us overlook, however, this proposition, and take the question from a practical standpoint. When an arthritis begins with a chill, whether it is single or multiple at the start, some one of the joints is going to be finally ankylosed unless it receives efficient surgical treatment from the beginning. If you read the textbooks you will find a description of acute inflammatory rheumatism as commencing without a chill, and one of the means you have of differentiating inflammatory rheumatism is that it is a multiple arthritis and begins without chill; that is wherein you differentiate it from an acute infective osteomyelitic lesion in the first stage. But you do not differentiate it by that from the metastatic type of arthritis that leads to ankylosis. In the metastatic type of arthritis which leads to ankylosis in the first place you have a large effusion in the joint, and you know it is an arthritis and not an osteomyelitis.

EPITHELIOMA OF THE NOSE

HISTORY

Male; occupation, farmer; age, fifty-two. Admitted December 8, 1911.

Family history negative; no malignancy. Returned to the hospital to be operated for a growth on the nose and one at the external canthus of the left eye. The patient states that he was operated on in 1903 for a carcinoma of the lip, which at that time was excised. In July, 1904, he was reoperated for a return of the growth, which was excised, and the inferior maxillary glands were removed. The growth returned for the third time and was again operated in June, 1906, when the tumor was excised. The return of the tumor occurred six months following each operation. In November, 1907, the lesion was again operated at that time. Very little tumor mass had reappeared. All of the preceding operations were performed on the lower lip. No return of the growth on the lip has occurred since November, 1907.

The present lesion started in 1899, and took the form of a small nodule. This was preceded four months by a trauma at the site of the present lesion from a blow. For the past twelve years the nodule has had scales form over it, which drop off and reappear only to again exfoliate. The lesion has had *x*-ray treatment, and has been cauterized many times, only to reappear in a few months. The patient also states that previous to his operation in 1903 he was accustomed to playing the jews'-harp, which irritated his lip so that scales would form, exfoliate, and then bleed. The slight lesion at the outer canthus of the left eye first made its appearance in 1905, and has not grown any in size since then. The only subjective symptoms attending either of these lesions is a slight itching. As far as he knows, the patient states that the lesion on the eye was not preceded by trauma. None of the lesions have ever discharged any pus, although at times a serous-like material exudes from the surface. The patient states that his general health has always been good.

COMMENTS AND OPERATION

It is seven years since we did the first operation on this man. We have performed three operations on him. After seven years, without evidences of a recurrence of the disease, we find that there has developed another carcinoma in the neighborhood of his eye. I am going to take a flap out of his arm and put it in there and it should live. This is the only place where we can do that. It is the only place where a flap will live this way. You can take an epithelial graft the full thickness of the dermis and put it on like a piece of cloth or a patch on trousers and it will take and live. One danger is the secretion from his eye, but I am going to protect this with collodion. His glands were taken out. He had no return there, but in the cheek.

In connection with the subject of grafting it is interesting to note that Tuffier, of Paris, is the only man who did ovarian grafting right. Morris, of New York, started it and lost out because he did not continue doing things. I received a reprint from Tuffier yesterday in which he reports a large number of cases of ovarian grafts—I think, 168. He takes out the ovary of every patient who has salpingitis, but he does not take it out and throw it away, but removes it and puts it back external to the rectus muscle with the hilum of the ovary turned in.

Cases of epithelioma of the eyebrow and the margin of the nose are the least malignant of all the epitheliomata we know of, and these are the ones I think that give the best results with the *x*-ray. As a rule, you get fairly good results in a number of these cases. These cases illustrate very well that nature is almost able to do the work itself, as with a tuberculosis of the lymphatic glands in children; if you will give nature a little boost, it will do the rest. This is the interpretation to put on that. Operating on the lid is unsatisfactory, but treating such cases with the *x*-ray has been satisfactory in some instances. I am suturing that in to immobilize it accurately. This is a tacking stitch to keep it so that it will not be elevated by the hematoma. I think this is the key stitch to this kind of work. It produces accurate apposition at the base during the process of repair. It is the same plan exactly that I adopted in the case I showed you yesterday. There is some oozing from the stitched wounds, but after the collodion dressing is applied it will stop.

PELVIC TUMOR

HISTORY

Female; single; occupation, teacher; age, forty. Admitted December 14, 1911, on account of an enlargement in the lower central portion of the abdomen

Family history: Mother died of tuberculosis at age of twenty-four; and father at sixty years, also one sister, all of the same disease. Had scarlet fever as a child. Has always been well since. Uses tea and coffee in moderation.

Menstrual.—Has always been normal, three to five days, five napkins a day until three years ago, 1908, when she noticed a slight decrease in flow, only flowing three to four days and four napkins.

Present Trouble.—On the fifteenth of April the patient states that, after retiring at night, she noticed that her abdomen protruded to an abnormal degree. For two weeks previous to April 15th the patient had been easily fatigued, irritable, and extremely nervous. Had also been troubled with insomnia. The patient has been in the habit of getting up to urinate four times every night. She has had no gastro-intestinal symptoms. Her feet are occasionally swollen on rising in the morning. On December 1, 1911, the patient became suddenly ill. She experienced a rather sharp abdominal pain. This pain was over the lower portion of the abdomen and lasted for several hours. She vomited twice after the onset of the pain, and had a slight chill one-half hour after the onset of the pain. She then states that she felt feverish. She called a physician, who told her that her temperature was 101.2° F. This pain finally subsided about two hours later, but she has not felt at all well since then—has felt feverish at intervals. Her appetite has been poor, and she has been occasionally nauseated, but has not vomited since December 1st. The abdominal swelling is slightly tender on pressure, and she experiences a dull, dragging pain over the site of the enlargement, chiefly noticeable on walking. The patient states that the next day after her sudden pain, which occurred on December 1st, the tumor was more tender and seemed to have increased in size. This, however, gradually got smaller until it had reached its former dimensions. It has been getting progressively larger, however.

Physical examination shows a large, smooth, freely movable mass extending from the symphysis to five fingers above the umbilicus and laterally two hand-breadths across the abdomen. The consistence of the mass is hard, and feels in spots as though there was fluid under tension. However, no wave of impulse is transmitted across the abdomen by this mass. There is no dulness in the flanks, and percussion of the abdomen over the protruding area shows dulness, not tympany. Urine examination, negative.

COMMENTS AND OPERATION

Dr. Murphy to Interne: Read that history about menstruation again.

Interne: The menstrual history has always been normal, so she states.

Dr. Murphy: What do you mean by normal? What does she mean by normal?

Interne: She has had a little dysmenorrhea in first day.

Dr. Murphy: That isn't the idea. What is normal for her? There is no fixed normal menstruation, you know, when you come to average the thing up. One may be normal with a six days' menstruation and one with a two days' menstruation. Whenever you are making a diagnosis for lesions in the pelvis it is of great importance to know this fact, and especially so in this particular case, because the woman has a large tumor in her pelvis, eight or ten inches in size, which she did not notice until when?

Interne: April 15th.

Dr. Murphy: All right; if she didn't notice it until April 15th, what relation did that growth bear to her menstruation if she has any? If this started in the uterus, she would have a menstruation five or six days all of the time. If, on the other hand, she has a cyst of the uterus, she might have a menstruation but two days. If she has a fibroid in the uterus, she would have menstruation probably increased, if it is a mucous fibroma, while if it is a subperitoneal fibroma, she would not have increased menstruation; therefore it is of the greatest importance in this case. Let the records show that she has a swollen tumor; it was impossible to make the physical examination because the hymen was covered, except under anesthesia. The cervix of the uterus is small, conic, and firm.

We rarely examine patients under an anesthetic with a pelvic disease, but here the conditions were such that we were unable to examine her without an anesthetic. It was impossible to make a bimanual examination. You remember the case we had the other day upstairs, how we made the examination beforehand, and then made a subsequent examination under anesthesia, showing the woman had a large tumor during pregnancy. We operated on her and found she had a collection of fibroids around the uterus which, had they not been removed, would have made delivery impossible, and we took them out. Having made the diagnosis, we made a large incision so that we could lift the uterus up out of the space; then, in place of tying the fibroids, we cut the peritoneum, slipped it down, and top sewed it with a fine needle.

Referring now to this case, I find the cervix runs backward and the uterus appears to rest on a tumor that comes in from behind and extends away up. This patient, being under nitrous oxid, is not relaxed; the abdominal muscles are tense and distended. I can feel something in the vagina, and the uterus appears to be quite tense. Her abdominal muscles are rigid. This examination is very unsatisfactory and gives me no light that is of value. Now read over that history again, so that we can, if possible, get these facts right. [History reread.]

Now, from this history and this examination I know positively that I have not been able to make a definite diagnosis as to where this tumor is, nor even as to whether it is a fibroid or a cyst. I know it is a large tumor, but I do not know enough about it. There is not enough in this history on which to make a definite diagnosis, and I accentuate this because it is irregular for us to do work this way. We believe that this can and should be made out by an examination, but I do not wish to put her under ether now and then go back to the gas after I have started it in this other way.

There you get an idea of the size of the tumor. This has a tendency to stay away up on the right side; it is rounded, hard—hard as a fibroid, either of the ovary or of the uterus. I recall one of the great surprises of my experience in a case once presented to us in New York by Dr. Edebohls as an ovarian cyst. He explained the case and said the tumor was over the promontory of the sacrum and looked just like an ovarian tumor. It really turned out to be a cyst of the right kidney. I never have gotten over the lesson that this taught.

There is the usual incision through the right rectus muscle—not through the muscle, but through the sheath of the right rectus muscle; then the right rectus muscle-fibers are all misplaced to the left side, and the peritoneum is lifted up as you see it here. We have to make a larger opening to go above the umbilicus here, as we cannot deliver the tumor otherwise. It appears to be a swollen tumor, as we described it before making the opening, and of great size. There are some adhesions above, and it is just as hard as it was before, and now I see shining areas on it. There are some adhesions in the upper right side of the abdomen. I will endeavor to locate it and see if we can deliver it now. We make a larger opening. Her abdominal muscles keep a little more tense than usual. It is a cystic ovary, or a fibroid undergoing degeneration, with a rotation of the pedicle, but not a complete rotation to the degree of strangulation. There is the uterus, as you see. It began in the uterus; there is the cervix of the uterus, and this is a fibroid, and it looks like a fibroid that is probably undergoing a sarcomatous degeneration. It originated in the uterus near the fundus. There is the bladder. You see I am amputating this [the uterus] in the regular way from behind the fornix, turning it out near the cervical canal.

There is the cervical canal. I am coming forward, keeping clear of the bladder, freeing it, leaving the peritoneum on as I push it up. We amputate the uterus in the regular way from behind forward. In taking out the uterus in this way it is merely a dome, and you have no more trouble in removing it than you would in removing an ordinary ovarian cyst. There is the uterine artery. See it! You let it bleed like you do the radial artery, then you know you have it. We will turn this around. There is the stump in the field all the time. We will commence this plan of closure of the cervix. We are sewing up the muscularis of the uterus, getting good compression with apposition, so that there will be no oozing from this tissue. You can see here the cut surface of the stump of the uterus. All this uterine surface is approximated and compressed. We will go to the other angle. I am cleaning up this canal. There is no bleeding. We will get the hemostats properly placed. There is the broad ligament you see on that side, held in position, and we will tie that into the crease made by the angiotribe. We will turn all this stump inward and protect the abraded surfaces completely and perfectly by this invagination, turning the raw surface down, so that we will have no exposed raw surface in the peritoneal cavity at the completion of this procedure.

Now, you can see, in analyzing this case, how important it was for me to know how many days that woman menstruated. Let us go back to illustrate the real value of it, because this thing started as a fibroid. It started as a mural fibroid. It escaped as a mural fibroid, which means that her menstruation should have diminished, instead of increasing, as the fibroid escaped from the wall of the uterus. If the fibroid had remained in the wall of the uterus, the duration of the flow and the quantity of it should have continued as time advanced, as is the rule, to which there are very few exceptions.

I am tying again, as you see, right into that crease. That takes care of this broad ligament. We will cover up this stump entirely, just the same as we do the stump of the opposite side. Those who are near can see we are picking up that tissue; I am going outside of the position of the ligature and am sewing the stump of the broad ligament in around the cervicocorporal portion of this uterus where we have amputated it.

In the clinical course of these cases you have a typical menstrual history that varies but very little. We will demonstrate to you our method of handling this stump. It is the regular way of handling the stump. You can see the stump very nicely. It is covered over between the two round ligaments, and these broad ligaments are used as material to cover over the raw surface, so that we will have no raw surface left there. We will drop these sutured ends. We will inspect the zone from which we have removed these adhesions. There is the zone there. This tumor was detached above.

Let the records show that the omentum became firmly attached to the exposed or denuded zone of the tumor. There is the raw surface. Here is the intestine, but the intestine was not united to it. We will tie these oozing vessels. This abrasion is on the omentum and on the under surface of the transverse colon. All this abraded surface of peritoneum should be covered and kept covered with one or two puckering stitches. You see, I have it now nicely protected. Let us clean out this side nice and dry. Pressure always means trauma. Trauma under anesthesia means depression and shock as much as trauma without anesthesia. Now, we are ready to close up.

We will commence to close the peritoneum in the regular way at the extremity of the cut surface. That is very easily accomplished in these large loose abdomens; occasionally, where one has a muscle tension on, it is not so easy to turn the edge outward. You will notice the divided edge of the peritoneum. It comes out without the slightest effort on the part of the operator. It leaves us with no denuded surface in the peritoneal cavity.

Let the records show that the tumor was solid, with areas of degeneration and fluctuation; that it began in the dome of the uterus in the muscular wall; that it gradually became eliminated from the muscular wall and became a subperitoneal fibroid; then elongated, partially rotated on its axis, that is, for about two-fifths of a circle, but not sufficient to produce a complete strangulation. At this time, or subsequent to it, a partial necrosis of the dome of the tumor took place; the wall was penetrated by a portion of tissue that had changed in consistence and color from the fibroid, resembling either a necrotic zone permeating through the wall of the tumor or a sarcoma developing in the wall or close to the wall, and producing adhesions to the omentum and lower surface of the colon. These adhesions were separated by the rubbing process before the tumor was removed and carefully inspected subsequently and the abraded surfaces left from them accurately covered.

Let the records show that Sister Victorine reports the sponges counted and correct.

There seems to have been a sudden change in the size of this tumor. The history is not of much value. There was some manifestation in the way of pain, some manifestation in the way of local irritation, and some manifestation of reflexes. When that tumor with the pedicle rotated on its axis two-fifths of a degree, in the manner that an ovarian cyst rotates three-fifths of a degree, or where the intestine does this, the rule is that there is a pronounced venous stasis—a beginning thrombosis. This produces pain, nausea and vomiting, local sensitiveness, elevation of temperature, and a leukocytosis. In short, it gives all the symptoms of an ordinary appendicitis due to an entirely different cause, and differing essentially from an acute infection by the pressure of a tumor early in the manifestation, and the only way in which it can be differentiated is by the presence of a tumor.

The first case of this kind I saw was in a patient in the second story of a house at the northeast corner of Harrison and Morgan Streets. It was on a Sunday evening, and I had another engagement and was in a great hurry. I was called to see this patient, and the doctor who had charge of the case said he had examined her repeatedly; that he had been treating her for an excessive menstrual flow, and she had no tumor previous to that evening. In the morning she had pain, and when I examined her she had a discernible tumor in the pelvis extending well above the brim. It was sensitive to pressure. She had pain, nausea and vomiting, soreness, elevation of temperature—the four cardinal symptoms of appendicitis and in their regular order. I insisted that it was not a case of appendicitis; that it could not be appendicitis with that tumor there; that the tumor was the cause of the trouble, and that it was probably a rotated pedicle.

Since that time I have had a dozen or more cases of this type of condition, leading to the same diagnosis because the symptoms were right, and overlooking the fact that a tumor was present. We made our diagnosis, had the woman taken to the hospital, and operated, and found she had a twisted pedicle of a tumor which accounted for her symptoms. Remember, with a twisted pedicle these patients get elevation of temperature. Wherever you get a thrombophlebitis that is acute from strangulation, without any evidence of pus infection at all, you get an elevation of temperature and a leukocytosis. This is due, we believe, to the thrombophlebitis which is produced. The tumor usually increases rather suddenly in size because there is a venous stasis without arterial strangulation. That is one of the essential things. That is why it swells. That is why when you have a hernia or knuckle of gut getting very large the venous current is stopped by pressure from this long before the arterial current is stopped. This also dilates the uterus.

The two conditions that can produce adhesions and cause a change in the conformation and appearance of the tumor are, first, strangulation, causing, when that rotation of the tumor takes place, a partial necrosis; and, second, a transformation of the fibroma into a sarcoma—a rather rare condition, but still an anatomic and histologic possibility and a clinical fact, though not frequent. The tumor that the patient should have after this secondary tumor is always a sarcoma, and never a carcinoma. Remember, if it were a carcinoma it would commence in the mucous membrane, never in the muscularis, because carcinoma always originates in either ectodermic or endodermic tissue, and never in mesodermic tissue, and the muscularis is one of the latter.

Carcinoma with fibroid is not at all common in this country. Bland-Sutton investigated the frequency of carcinoma in the

London hospitals, and found that 4 per cent. of the uteri that were removed in the London hospitals had carcinoma of the mucous membrane. That, I believe, is away in advance of any investigations we have made in this country, and I believe is easily explainable on this basis, that the fibromata in England are not operated on as early as they are in America. You recall the evolution of the operation of hysterectomy for fibroma of the uterus. When I was beginning to do this class of work these were the indications: (1) A patient having a hemorrhage which was hazarding life; (2) a tumor pressing upon some of the neighboring structures; (3) a tumor that was undergoing necrosis. Otherwise, a fibroma of the uterus should be permitted to remain. Those indications were laws at that time, and they prevail yet in many European countries, while in this country we have taken up the plan of operating and removing fibroids in all patients who are in the menopause as soon as they are discovered, because then they are more easily removed, and operations give splendid results, and it is no more dangerous to remove a fibroma of the uterus than it is to remove an ordinary cyst of the ovary. In Bland-Sutton's cases in the London hospitals the patients who were operated on were very much older on an average than those upon whom we operated in this country for the same condition. I regret he did not bring out this point.

There is a division of this tumor, as you see [showing it divided]. When you come to look at this tumor, the question arises, is this a sarcoma or a cystic change that has occurred in a large pedunculated fibroid? Here we have a beautiful mental picture of the uterus. There is no definite line of demarcation between this part of the tumor that is changed and the remaining portion. There is no encapsulation. There is no line of separation. There is still a further advance in cystic degeneration, and it is my interpretation now, from the gross appearances, that it is not a sarcoma—that it is merely a change the tumor was undergoing on account of deficient circulation due to the rotation of the pedicle. There is no line of demarcation between these two surfaces.

Here is another fluctuating mass deeper down that is in a

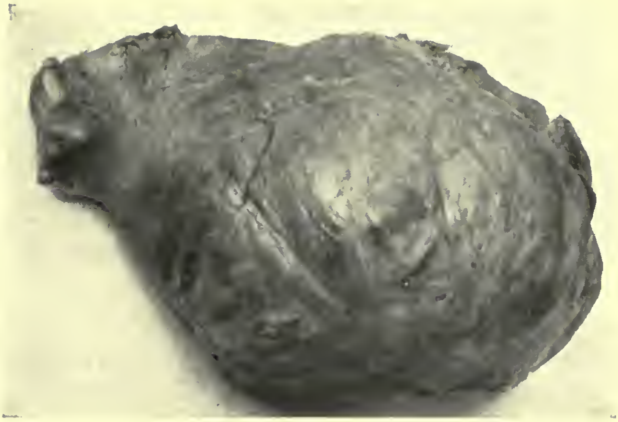

Fig. 7.—Pelvic tumor.

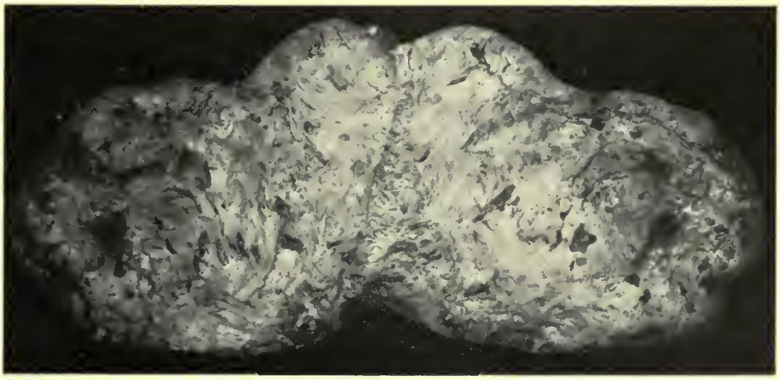

Fig. 8.—Showing divided section of pelvic tumor.

further stage of degeneration. That is the one I felt. It looked transparent when I began to open. There is cystic degeneration. These are connective tissue lined, never epithelial lined, because they are of connective-tissue origin. This is a very pretty specimen, illustrating all the conditions that develop in a fibroid from its normal region of vitality. As you see, there is scarcely any change at all in the lower portion, and it is not soft or fluctuating. There is the uterine tract, and here was the pathologic condition near enough to the uterine mucosa to have influenced her menstruation. We can say, after we have completed this operation, that it is probably not a sarcoma; that this projection from the uterine wall is due to the arterial or necrotic changes the result of rotation of the pedicle. This was an excessively long pedicle mass, as you saw. It allowed the uterus to rotate with the tumor, causing strangulation of the venous circulation and retention of the arterial circulation, giving a soft, boggy mass. When the strangulation involves the arterial circulation, you do not have a soft, boggy uterus, but the dry type of necrosis and gangrene.

This is a beautiful case for a word-picture of all these changes, and that word-picture must be simply the annals of the sick. The history must be unvarnished and uncolored. If you write a story of a simple thing, be sure and get the facts, and not let the patient skip a year or two in giving her history. Then you will have a story from a symptomatologic standpoint that will correspond with the clinical findings in the enormous percentage of the cases.

Since Trousseau's time, no man has written a good book on internal medicine—a good book in the sense that no man has gone into a careful analysis of symptoms, as he did, from his observations and investigations of patients, because we have all endeavored to short-circuit to a definite pathology. Trousseau's word-pictures of lesions, although he knew nothing about pathology, are just exactly as good today as they were in his time, and if we could get some man that would write the histories of cases on the Trousseau plan by observation and attach them or make them a part of the pathologic findings of the present day, we would have an ideal medical book. We would have a medical book that would be a guide and a great assistance to the physician at the bedside; and then, if one could attach to it the report eliminating and confirming the facts that come from the laboratory, you would be able to arrive at a diagnosis with much greater certainty than if you were to pick out the pathology and try to make the laboratory findings correspond with your interpretation of the pathology, or if you were to send a strand of the patient's hair to the laboratory and expect the laboratory man to send you a definite diagnosis. I put it in this ridiculous way deliberately because that is what is expected from the laboratory today. That the laboratory is an aid of the greatest value we know, but it is like other things, fallible, and it is only *one* of the elements that go to make up an accurate diagnosis.

NERVE ANASTOMOSIS

The Musculospiral Nerve

HISTORY

Female; occupation, housewife; age, twenty-eight. Admitted November 2, 1911.

Family history: Grandmother died of cancer. No tuberculosis in family.

Married six and one-half years. Born and always lived in Minnesota. Habits good. Had all of the children's diseases. Scarlet fever at age of twelve. Complete recovery. No typhoid, diphtheria, or pneumonia. No jaundice or rheumatism. Occasionally attacks of tonsillitis. Curetment June, 1911. At age of seven had small tumor removed from left arm. No severe injuries previous to present trouble.

Menstruation began at thirteen, regular twenty-five-day type, each period lasting five to seven days; flow scanty. No pain; no clots. Occasional vaginal discharge; no odor. No burning on urination. Three children, oldest six, youngest six months, all living and well. Labor normal. In second labor had mastitis, chills, and fever. Curetment after last child. Does not think she had chill or fever, but had hemorrhage. No miscarriages. Occasional vaginal discharge. No odor or pain on urination. No history of specific disease.

Present Trouble.—Dates back to August 29, 1911. At this time patient was injured in automobile accident. She was thrown into a barbed-wire fence. Wire cut across left cheek and upper lip and part of right cheek. This extended through antrum on left side. Both bones on both forearms and tendons cut. All wounds became infected and discharged pus, especially on right forearm, when infection took place and no union. This is the fourth operation since her accident. Perfect union now of bones of forearms.

COMMENTS AND OPERATION

We have had this patient gone over by Dr. Mix to determine whether there was a paralysis of the musculospiral nerve. We thought this was a mighty easy thing to determine, and it would be if we had the forearm in a right condition, but the forearm has been cut by barbed wire. The bone has been divided and the extensor tendons cut. She has some extension of the fingers, but it is an extension of the lumbricales, with an extension of the short tendon of her pollicis. We believe this has escaped injury. She extends the thumb and fingers in a peculiar way, which is the type of extension we see in these cases. Here, you will see, we have to correct the deformity and restore the triceps. We have endeavored to find the musculospiral nerve in that mass of cicatricial tissue, and this is not an easy matter.

Those of you who were here when I was working out the nerves in the case of a man the other day, with all of his anterior group divided, will remember what a digging operation we had to do to get them free. We did so, finally, established end-to-end union by applying a flap for the extension of his three nerves—the median, musculospiral, and ulnar. That, you remember, was another type of injury. He was struck by a falling mass of iron that cut the arm across.

In the case before us we must go in back of here where the wire caught her and in through the cicatricial tissue. Before she was anesthetized I could not make out a neuroma. With the nerve divided you can palpate a neuroma above and below the scar. In this case I could not make out a neuroma. All these muscles are cut across and fixed so that we have nothing to go by. There is no response from the electric current. The cut was made just a little bit high, but it was a barbed-wire cut. It sawed through, and you do not realize how it pulled these tissues out. You see, it almost cut the arm off, cutting both arms and both bones, cutting this arm with all the extensors as well, and the other forearm.

We restore the triceps muscle. That is proposition number one. This patient's arm must come up so that I can get it in a little different position. You see, coming out as this does, it is very close to the groove. That is the exact anatomic line. The division of a muscle in this way is peculiar, depending upon the exact location in relation to the tear. When a muscle is divided nearer the head than the neck, the rule is that there is full restoration of function, with the gap filled up by fibrous tissue which connects the muscle ends. When a muscle is united above and below with fibrous bands, it is strong, and is as capable of sustaining weight as the original muscle was. There was a gap of three-quarters of an inch here originally. The muscle-fibers are still intact. The scar inside seems like a straight scar. It does not seem to go crosswise over the arm, such as the skin scar seems to go. I am following right close to the cicatricial line and freeing the muscles. I want to find the normal triceps fibers as near as I can in this cicatricial tissue. This looks like the nerve, but it appears to be rather too superficial for the nerve. It is spread out up here, and I think it is the aponeurosis of the muscle. Remember, the nerve is right down close to the bone. This is [indicating] the central fiber of the muscle. Here is where it ought to be in the scar tissue. It should be along here. I do not yet feel any evidence of a neuroma in this mass of scar tissue. There is the nerve-trunk showing up clearly and distinctly, so that we must follow it. There is the neuroma on the distal end showing up nicely. We should get this connective tissue out of the way. There is the nerve. Let us find out what was interposed between it. There is the nerve-trunk. You see there is inhibition of the nerve. See the artery above! There is no response to the electric current. Some of these fibers should be intact, but they are held down by a mass of connective tissue.

Let the records show that the nerves above and below the scar line were rather readily exposed, and this nerve was found to be entirely divided and the ends down in the scar tissue where the periosteum was torn through. Both ends were freed by careful dissection and separated from the periosteum for an inch in both directions from the line of division. There was apparently a fragment of bone taken out at this point [indicating] with the barbed wire. There was considerable retraction on the nerve, and it was freed and brought down by mild stretching.

The next thing is to get union where we have complete and perfect axons, and that nerve must be divided where there are living axons, living Haversian vessels. We have here the nerve exposed. There is the central vessel. We must see how far we will have to go on the proximal side, and I want to go as little distance as possible, so as to use the neuroma as a supporting material. I have not detached the neuroma on the other side. By going a little further back I will be in a proper position. There are the fasciculi sticking out everywhere. See them! We wish to put that nerve away from the bone where it will have the structures brought parallel to the long axis of the nerve, bringing the axons toward each other and keeping the nerve from contacting with the periosteum. There are the muscle-fibers. Here is the penetration. Here is where I am going to make my encasement for this nerve.

You remember the beautiful experiments of Levings, of Milwaukee, in which he showed the value of having all muscle-fibers parallel with the axis of the divided nerve. He showed that if you did not have interposition of loose connective tissue from the outside, you would have fairly good conditions for regeneration of the nerve.

I am going through the trunk. The axons will approximate each other unless there is connective tissue interposed between them. If I did not have this encasement material I have here, I would not do this operation in this way. I would make an extension of that nerve, but having this splendid encasement material, composed of the aponeurosis of the central fibrous strands, or the central fibrous strands themselves, I am doing the operation you see. These are the contacting axons from the proximal to the distal side.

Let the records show that the end of the nerve is united at the central septum to the triceps muscle. You see, as I pull the triceps over it fully encases the nerve. The fibers of the muscle are running in the direction of the long axis of the axons of the nerve. I am reëstablishing the triceps muscle over the crease.

If this muscle were cut in the center, the fibrous tissue would develop, giving it its tone and original strength.

We had before us an illustration of this a short time ago in an athlete, who made his living through athletics. He had his biceps muscle cut across in a railroad accident, it being cut clear down to the bone. As a result, there was a depression in his upper arm large enough to admit my fist between the ends of the bone. That depression represented a connective-tissue mass, uniting the divided ends of the nerve. At the time the railroad company wished to settle with him for the accident, but he declined. He declined on the basis that he did not know how much power would be restored in the muscle. He said that if the muscle power was restored, he did not want any payment from the railroad company except for the loss of time. If it were not restored, the railroad company would have to pay him a compensation sufficiently great to provide for him in case he could not earn his livelihood. The railroad company's decision, on the advice of medical men, was to the effect that they would accept his proposition. He was delighted at the prospect. First, he was delighted that anybody should have confidence enough in the restoration of his muscular power to make that kind of settlement, and he felt that if railroad companies had as much confidence as that, he had every reason to hope that he would get a good result. He had a full and complete restoration of the power of his biceps. He could lift exactly the same number of pounds that he could before the accident with his biceps, showing complete restoration of function of the biceps after its division, and that is exactly what should have been expected from that case.

Coming again to the case we have here, we unite this so as to get good apposition over all the area where the muscle was divided, and contacting the fibrous tissue after freeing it from the bone. What prevents a good result in these cases is the fixation of the muscle to the periosteum, which does not permit the muscle to cut across the line of division. The lower segment of the muscle, when it is bound to the periosteum, must be free from the periosteum, as we do it here, and end-to-end union made of it.

You can see, I have filled up that gap fairly well, and I have a contacting arm. We will have practically no depression in this arm. Let us talk about the nerve element in this case. What will be the outcome of the nerve? Some years ago we showed definitely what could be expected in every nerve division. We showed that the nervous system was divided, so far as regenerative power was concerned, into three main classes of nerves: First, the cerebrospinal group, of which the musculospiral is one; second, the sympathetic group; and, third, the nerves of special sense. We showed in that article that every nerve, if approximated in the proper way, is capable of regeneration, providing it has a neurilemma in its makeup in the construction of its axons. We showed that the axon was made up of an axis-cylinder or central transmitting zone; that the myelin sheath was the insulating zone, and the neurilemma the *regenerating* sheath. Every nerve that has a neurilemma has in it the potency of regeneration, provided the ends are brought in apposition without the interposition of foreign material, and included in this foreign material in the nerve is a mass of connective tissue. Connective tissue is the greatest barrier against the reunion of nerves.

You have then the entire division of the nerve. You have the divided ends of the nerves of special sense. The axons of the nerves of special sense have no neurilemma; as an instance, the optic nerve has no neurilemma. It has a medullary sheath and an axis-cylinder. The nutrient cell body is in the retina. It regenerates, if it regenerates at all, from the direction of the retina. It does not regenerate at all because it has an axis-cylinder, has a medullary sheath, but *no neurilemma*. Therefore, when the optic nerve is divided, no matter how accurately you approximate it, it never reunites. The same is true of all nerves of special sense—the olfactory, the gustatory, and the auditory. Once they are divided, they are permanently put out of commission. Why? Because the axons have no neurilemma. The sympathetic system has its axons made up of an axis-cylinder, no medullary sheath, and a neurilemma. It is, therefore, capable of regeneration, that is all of the sympathetic nerves, and they are incapable of preventing short-circuiting in a large percentage of the cases, and that is why we have this cross-circuiting sometimes through the sympathetic nerves. In the first place, they short-circuit to the ganglion, and, second, through the trunks, because they have no myelin sheath, which is the insulating sheath.

We have next the nerves in the spinal cord and the nerves that are attached to the spinal cord, as well as the ordinary nerves of motion which originate in the cranium and supply the muscles of the face. The nerves of the face are exactly the same as the spinal nerves. They originate always from, and have their continuation in, the spinal cord. I am speaking now, of course, of the axons of the lower motor neuron, and not of the upper motor neuron. These axons all have a neurilemma from the time they leave the spinal cord in the motor nerve, and from the time they leave the spinal ganglion in the sensory nerve, and from the time they leave the cranial foramina in the facial supply. Therefore, you have these nerves all with a neurilemma, both the sensory and motor portions, all capable of regeneration after division. When you have a division of the facial nerve, if you recontact the ends, so that the bundles of fibers come in apposition, these necessarily develop axons through the regenerative potency of the neurilemma, the axon coming from the proximal side and extending on down to the peripheral or terminal end. When a spinal nerve is divided in any part of its course external to the posterior root of the ganglion, it is capable of regeneration, because it has its axon, it has a neurilemma from the time it leaves the anterior commissure of the cord in the motor zone, and has a neurilemma from the time it leaves the posterior ganglion in its sensory axons.

The period of time that may elapse between the time of division and the time of approximation of a nerve, and still have its regenerating potency effective, has never been ascertained. The longest period of time that has elapsed, that is, in our case, from the time of division of a nerve to its restoration, was twenty-six years. From that time we can conclude that there is no period of time that is sufficiently long to positively inhibit or prohibit the regeneration of the distal portion of the axon after a systematic and scientific reunion of the nerve-trunks.

Immediately after the division of a nerve-trunk you have at once the separating and continuing on to final death of all the axis-cylinders, the neurilemma, and the myelin sheath except the nucleus of the myelin sheath cells. These nuclei first swell, multiply, reproduce, and you have synchronously with the act of degeneration the beginning act of regeneration in the particular part played by the neurilemma in the regeneration of the cell. In the process of degeneration the first thing to break down is the axis-cylinder. It commences to fragment about the fourth day. Up to the fourth day you have transmission of the faradic current to the terminal end. Why? Because the axon has not yet fragmented. By the seventh day the axis-cylinder has completely fragmented, and transmission will have ceased. Synchronous with the breaking up of the central axis-cylinder you have fragmentation and breaking up and fatty degeneration of the myelin sheath, swelling, enlargement, and death on the margin of the cells, elongation of the cells, and the fragmenting of its nucleus, and about the eighteenth day you have these cells of the neurilemma enlarging and contacting one after another, so that it makes a series of cells extending from the terminal filaments of the nerve back to the first node of Ranvier on the proximal side of the line of division. That first constructs a new series of neurilemma cells. Then that process of myelin substance, that tube of myelin substance, is ready for the extension of the axis-cylinder from the proximal side as soon as the apposition is made. In other words, if there is a distal regeneration of the neurilemma and of the myelin sheath before approximation is made, the nerve-trunk retains its full and normal size all of the time, but there is no axis-cylinder regenerated in the distal portion until the nerves are again actually contacted, and these tubes of myelin substance surrounded by neurilemma cells are the tubes through which the axis-cylinder grows nearer to the end. Whether it be a divided sciatic with the lumbar below, or a divided median, or a divided ulnar, with only a part of it below, it regenerates to the full extent of the nerve.

The next element you have in connection with trauma of the nervous system is the division of the spinal cord. The spinal cord above the caudal zone is made up, both in its motor and sensory areas, of axons with a central cylinder and myelin sheath, but no neurilemma. Therefore, when the spinal cord is divided or any of its axons are divided above the cauda, there is a complete, permanent, irreparable degeneration of the distal portion of the sensory or motor axons and complete degeneration of the sensory axons back to the ganglion-cell of the posterior side. When the cord is once cut, regardless of the cases reported in literature, and regardless of the cases that have been reported from an experimental standpoint, and regardless of the cases in which the musculospiral has been divided, there is no regeneration of an axon that has an absent neurilemma in any of the types of animals or in man that we know of. There are many misleading experiments reported in connection with the division of the spinal cord of frogs. A frog's cauda equina extends up to the spine of his scapula. His conus is at his scapula. If you divide the spinal canal below that, you divide the distal axons that have a neurilemma clear back to the entrance of the motor nerve into the anterior commissure of the cord, and, like the human cauda equina, if you approximate the nerves of the cauda equina after division, they grow together just the same as the sciatic, the tibial, radial, or the ulnar, because you are in the neurilemma zone of the nerve, and you have a full and complete regeneration of both its motor and sensory axis-cylinders and axons.

Now we come to the application of these elemental principles to this case. Here we have divided the nerve. Here we have it separated by a span of connective tissue. Here we have the ends rounded off and insulated by the neuroma or fibrous tissue. In sewing nerves together you must cut off all of that mass of connective tissue back of the position where you see the individual nerve fasciculi sticking out and uncovered, so that they can be directed toward each other, or brought in apposition as we prefer to have them when they are directed toward each other, and supplied by muscular fibers. Nerves of the upper extremity expand a greater distance when separated than those of the lower extremity. The one that expands the longest distance of any possibly is the fifth. There is a definite or well-established principle or law that a nerve is capable of regenerating in proportion to the size of its ganglion. The largest ganglion in the body is the Gasserian ganglion; the greatest potency of regeneration is represented in the trifacial or fifth pair of nerves, where you can take out an inch and an inch and a half, divide it, split it, cut it, sew it, still it grows. That applies to the axons of the fifth pair of nerves, and still the axons grow.

DUODENAL ULCER

HISTORY

Female; occupation, housewife; age, forty-seven; married; admitted November 27, 1911, because of vomiting of blood and passage of blood in stools.

Family History.—Father died of heart trouble after an illness of two years at age of seventy-five. Mother living, but has always had a poor stomach. Two brothers died of tuberculosis at the ages of nineteen and fourteen. One living brother has tuberculosis.

Past History.—Had the ordinary diseases of childhood, but no serious illness until fourteen years ago, when she had both ovaries removed for a cystic growth. She has for the past twenty years had a feeling of fullness in epigastrium after meals. Drinks coffee, but no alcohol.

Menstrual.—Menstruation normal to age of twenty-one, when she was married. Since that time, until her operation for the removal of her ovaries fourteen years ago, she had a great increase in the flow, lasting eight to ten days, and during the year preceding the operation she flowed about seventeen days of each month. She has had leukorrhea since ten years of age. No history of specific disease. Has had four children, all normal deliveries. She had eight miscarriages that she knows of during the twelve years between her marriage and the removal of her ovaries. These miscarriages all occurred after the birth of the first child, and she was two months pregnant when they occurred.

Present Trouble.—For the past twenty-four years the patient has had a heavy sensation in the epigastrium, coming on immediately after eating. This has been accompanied by a bloating and belching of gas and a throbbing in the head. This sensation reaches its maximum one to two hours after eating and is relieved temporarily by drinking hot milk. Sodium bicarbonate also relieves the symptoms. She has frequently had palpitation of the heart, coming from the pressure of the gas. She has always had a poor appetite, and has to take medicine always to obtain a bowel movement. On the fourteenth of November she first noticed that her stools were very dark and reddish in color. Had had no medication. On Friday, November 24th, ten days later, she had a feeling of nausea and gas in the stomach. This was accompanied by a weakness and dizziness, and she became very faint, and half an hour after the pain started she vomited a quart of black coagulated blood. This vomit also contained a few streaks of bright-red blood. The next day the stools were dark. She had not vomited before this time and has not vomited since, although the stools have continued blood stained. In the past four weeks the patient says that hot drinks burn her stomach and she feels as though the skin were off on the inside.

COMMENTS AND OPERATION

In analyzing gastro-intestinal cases you take a certain definite series of symptoms and arrange them in a definite way in order finally to get at a clear conception of the results.

You remember, some years ago, we outlined a syndrome in connection with intestinal obstruction. It consisted of pain, nausea and vomiting, borborygmus, and inability to get a bowel movement by the ordinary means—in other words, coprostasis. You will remember later we outlined a syndrome of symptoms in connection with appendicitis, which consisted of pain, nausea and vomiting, local sensitiveness, elevation of temperature, and later to these we added leukocytosis, always in this given order. When the order varied, we always questioned the diagnosis. If the temperature came first, we knew positively it was not appendicitis.

We have had now nine cases of typhoid fever with typhoid ulcers in the appendix. The first three were operated, these having been diagnosed as appendicitis, and ulcers were found. The last six we did not operate on because we made a diagnosis of ulcers in the appendix, giving a syndrome of symptoms, because in each of these cases we had temperature preceding the attack of pain. This is the order of symptoms which is so important. When you come to analyze cases of gastro-intestinal lesions, and particularly lesions of the stomach and duodenum in connection with the lesions of the gall-bladder, the three common lesions of the upper quadrant of the abdomen, you must take into the estimate certain things: First, pain. Second, and only very little less in significance, gas eructations. Third, emesis and then hematemesis, or the passing of blood in the stools. Fourth, the local sensitiveness on pressure over the ulcer zone. Fifth, distress or relief produced by the ingestion of food. These represent the five elements that must be weighed in making a differential diagnosis as to whether it is a gall-bladder lesion, a duodenal ulcer, whether it is a gastric ulcer, whether it is a reflex from the appendix, or from the pelvic zone, or from some other place, or from the central nervous system; that is, syphilis, which gives its gastric crises. All these, if they balance, if they come with each symptom in an almost uniform way, the case is then typical. On the other hand, if they vary, then you can put a large interrogation point on the abdomen before you open it and cut through it to the diagnosis. This is the best way for arranging that in one's mind to arrive at conclusions in making a positive diagnosis.

This woman has a history: first, of pelvic trouble extending over a long period of time. She has a history of uterine hemorrhage, which, in all probability, was a fibroid hemorrhage. She has a history, further, of prolapse of the uterus. All of these things could have produced in her the earlier symptoms which she had of lesions in the upper abdomen; but when you come to the later symptoms, the symptoms of her stomach attacks, pain followed by fainting, followed by hematemesis, then you know that you are out of the zone of reflexes, and you are in the zone of actual lesions of the duodenum, or stomach, or liver. Remember, when you have hematemesis with certain lesions of the liver, particularly the early stages of cirrhosis, and again in the very late stages of cirrhosis of the liver, we have hemorrhage. These hemorrhages must not be confounded with the active hemorrhages that come in connection with gastric and duodenal ulcers, notwithstanding they may be profuse.

I recall one of the first cases of that kind which I saw within a few weeks after I left the Cook County Hospital, at 116 Mather Street. I remember it very distinctly because I felt the man was going to die right then and there while I was in the house, on account of the quantity of blood that was being poured out. He had enlargement of the liver, sensitiveness on pressure. He had been a hard drinker, and had ceased drinking only shortly before the time of the hemorrhage, and the hemorrhage was explained by that observation. The case was not diagnosed as one of gastric or duodenal ulcer, but as a cirrhosis of the liver in the early stage. He reformed because he felt he was going to die. However, he lived for many years and I used to see him years after.

We have in the acute cases two types of ulcer of which you must never lose sight. One is the small, penetrating type of ulcer which is a little larger on the end than a lead-pencil; it bleeds intensely, and is not palpable from the outside of the stomach. The other is the large round ulcer in any stage of its evolution or repair. The most common type of ulcer that gives hemorrhage is the large round ulcer; but the acute, severe cases are the ones that have the small type, and they are the most dangerous, and many times are associated with all the symptoms of gangrenous gastritis. I had a very interesting experience of this kind in my early professional work, in the person of a doctor from Appleton, Wisconsin. He had all the symptoms of a large round ulcer of the stomach. He came down to Chicago, was seen in consultation by Dr. Billings, and we agreed that his case was one of gastric ulcer. He went away for a long vacation and the ulcer got perfectly well. He gained in weight from 119 pounds, which was his weight at the time of the consultation, to 156 pounds, and resumed his practice feeling perfectly well. Some time after, in the last part of

December, if I recollect rightly, he had a sudden pain in his abdomen; he began to vomit; he had a chill; he had elevation of temperature up to 104° F. He became delirious, and he had blood in his stools continuously, and he went on and died in three weeks. The diagnosis was that the old ulcer had finally taken on a new and wicked form: that it had taken on a severe destructive type of sepsis or gangrene, and led to the fatal termination.

At the autopsy, when we opened the abdomen, nothing at all could be seen from the peritoneal side of the stomach except the seat of the old ulcer; this was of a white, pale appearance that comes in connection with an ulcer that has repaired, and it was clearly discernible from the outside. Over in the pyloric zone there appeared to be a congested condition. The vessels seemed dilated, but nothing could be felt. The stomach was opened, and we found the real cause of death was the type of ulcer I have described. He had eight or nine perforating ulcers of his stomach; these extended down to the blood-vessels, and he was bleeding from them. There was an area of necrosis in the neighborhood, and that all led in the direction of the blood-vessels, which accounted for the hemorrhage.

Notice how cautiously we are handling this, because it is only four weeks since she had a hemorrhage. There is your pathology. You see there is an acute infiltration all along. There is the duodenum, which is adherent to the gall-bladder. There is no exposed duodenum here. I want you to see how nature protected the ulcer. She has filled in the omentum and covered the whole thing up. It is beautifully illustrated in this case. There is the thickened zone in the duodenum. The ulcer is just outside of the pylorus. The glands are quite markedly enlarged. I am going to feel the gall-bladder and see if we have secondary impairment of the gall-bladder or if we have gall-stones in it.

Let the records show that the gall-bladder is compressible; that there are no stones in the cystic duct.

Now I will come out in the direction of the head of the pancreas. Here is the head of the pancreas. It is harder than normal. It is not woody hard. It is not the type of woody hard you get with a pancreatitis, as I showed you some two weeks ago. The ulcer extends fairly well down toward the head, and there is a crater in the duodenum, at the head of the pancreas, on the inner side, where the ulcer shows. Outside of that there is some induration. The pancreas is primarily the seat of disease; the ulcer is in the duodenum on the duodenal side, accompanied by adhesions. What we wish to do is to turn the food current out of this field into another field. This is one of the unpleasant cases to operate on because of this enormous deposit of fat in the mesocolon, which greatly hampers the procedure. We are in the lesser peritoneal cavity, and I will spread this tissue downward so as to let the stomach come well out in the greater peritoneal cavity and into the field that is important and to get our line of union, which is here. One never likes to operate on a patient with this large deposit of fat in the mesocolon. Such patients do not stand operation very well.

I am drawing now the lesser peritoneal surface of the stomach out into the greater peritoneal cavity. You see, I am drawing out here so that it will show in the greater peritoneal side of the mesocolon, pulling this portion of the stomach out. The closing of this lesser peritoneal cavity is important, first, because it throws the surface of the stomach where we desire it. Second, it is important because it prevents the stomach from receding into the lesser peritoneal cavity after the operation is completed, and does not permit this edge of the divided mesocolon to interpose itself between the lines of union. It is important, in the third place, because it does not permit of a lesser peritoneal cavity hernia. When the lesser peritoneum is closed, the intestine cannot get under so as to cause a hernia. There are some fatal cases of this type reported in the literature. We pick up the stomach and find the origin of the jejunum and get the ligament. There it is, right there [indicating]. You know that is the origin. We simply let the omentum go back in. We rotate the stomach out again, so that it can stay in the field. So much of the stomach is out of my field, and I must keep it out until I get through.

Now, we are ready to make the anastomosis. We will do this with the button. In putting the needles in this way, you put them in parallel to each other. This is Pagenstecher linen. You keep the needle in and divide the intestine between the needle. Formerly, we used to pull the needle through and then occasionally cut the thread in dividing the intestine. This is the Hartman clamp, worked out by my former partner, Dr. Hartman. We cut the thread short up to the knot, and have the knot on the end. One of the greatest condemnations of the button was received from a case of Dr. Marvin, who had the end of the string hanging out over the button. It was found there at the postmortem examination. He reported his findings, which gave us a chance to estimate what had happened. He did not realize, after he had published the facts in the case, what he had done.

We do this [indicating] the same way as we did the other. We go through all the coats in and out, four or five times, depending upon the length of the tissue, and draw the needle through and retain it there. We do the same way with the second needle. In putting in this button you catch it on the cup and then put it in. With the other you catch it on the cylinder. You cap on the cuff and get it in, then change it and take the cylinder. Here is the other half of the Hartman forceps. That is pushed down on the button below the cap. Now we are ready to make the approximation. We will take off the clamp from the jejunum, so as to allow the jejunum to come around in the position necessary, and you see the opening is closed behind with my thumb.

Now we are going to put in stitches in such a way that there will be no traction on this zone of approximation. We do the same thing when we use a suture. We take off tension from the line of fixation. You will see there has been no possibility of any type of escape of material from this zone in which we were working. You see here the advantage which the button has over the line of suture in that particular. We do probably three-fourths of our cases with the button. Occasionally, when we have two cases in a day, we do one with the suture and do the other with the button. We can do both. We like the button better than the suture because we think it requires less skill, less care, and that it is no additional hazard in any part of the gut.

You will recall the number of buttons that were not found after operations, and it was believed they were retained in the intestinal tract. In rebuilding one of the hospitals they found 21 buttons at a sewer junction, showing how perfectly regular they were in their passage.

In making this anastomosis you noticed that I made it so as to have traction in the same line of axis propelling the contents of the intestinal canal. You see, it was put up in this shape [indicating]. There was a normal relationship established between the jejunum and the stomach. In the upper zone of the abdomen it is much more important to have an ectropium of the peritoneum than it is in the lower.

In weighing the statistics with the button and the various means, particularly the suture, which is probably the only one that is lined up with it at this time, we have to consider, first, what are the dangers with any method of approximation? The dangers with any method are—first, failure of union, which, of course, is very slight with any of the methods that are in vogue at the present time; second, and more important than the danger of failure of union, is the danger of hemorrhage. The greatest mortality following gastro-enterostomy at the present time is from hemorrhage. What are the dangers from hemorrhage with other methods, and what are the dangers from hemorrhage with the button? With the button it is practically impossible to have a primary hemorrhage. Why? Because the blood-vessels are compressed in a circular manner when you press the button taut. Second, with the suture you have blood-vessels crossing the field, and with the continuous suture, which is in the mucous portion of the gut, you have to secure it from oozing or bleeding around it. But from the reports of cases you know that surgeons do have hemorrhages and sometimes they are serious. At other times they are not serious. Sometimes these hemorrhages are sufficiently severe to require reopening of the abdomen.

Failure of union is very rare with any method where proper respect is paid to the application of the method used in making the union, namely, if the sutures are properly placed; even though they penetrate the entire wall of the abdomen, they do not give a leakage of septic material that interferes with union, and that is particularly why we are using Pagenstecher linen. This linen can go through the entire wall of the intestine, and, having no capillarity, it does not conduct infective material along it. Ordinary silk is rarely ever used now for this purpose without filling it with paraffin or some other substance. Paraffin silk or coal-tar paraffin silk is as good as Pagenstecher linen, as silk treated in this way has its capillarity destroyed. Moses Gunn used waxed silk for all these cases. He used it for drains and for suture material when I began to study medicine. He did not know the reason why, but he was a keen observer of facts, and he knew that he secured good results, and these are the final tests of any material.

Failures of union are due sometimes to different conditions. First, failure of response on part of the tissue to react in a general way to the mechanical pressure of the suture or the mechanical pressure of the button. Second, to the presence of infective microörganisms at the point of contact. This last is the most potent factor in the production of non-union, and it matters little what the material may be, when these microörganisms are present there is likely to be a failure of union, not as great a certainty of failure of union as there is when you have infection in the skin, but there is a likelihood of failure of union because we know the peritoneum endeavors to coffer-dam and overcome every type of infection, but sometimes there is a failure of union in spite of this. Let the records show that Sister Laurie reported the sponges counted and correct.

The immediate results in gastro-enterostomy now show about 1 per cent. mortality. The final results, looking to the relief of the symptoms for duodenal ulcer, are, on the whole, very good, and very good without excision of the ulcer; that is, we get a larger percentage of relief of the symptoms following gastro-enterostomy for duodenal ulcer than we do for gastric ulcer—

decidedly larger. Duodenal ulcer is occasionally excised. In a duodenal ulcer, encapsulated as this was by adhesion, by suppurative conditions, you have as a result a periduodenal inflammatory condition. If you separate the adhesions and expose the wall of the duodenum, you will have an indurated wall. You will have a wall with no endothelial cells on it, and you will have unfavorable conditions for union, and no one would think of making an excision of that ulcer for its permanency of cure when the chances of cure are better without dissecting the ulcer.

This is a figure-of-8 stitch. The lower loop includes the aponeurosis of the recti muscles; the upper includes all the structures above the aponeurosis. That gives you union by a suture with which to hold together the wall that is important for maintaining a permanent support in this abdomen at a point at which it was divided. No matter what happens, the silkworm-gut stitches are there to stay until they cut or are removed. We get support for twelve or fifteen days, because it takes that length of time for pressure necrosis to occur in the white fibrous tissue that makes up the aponeurosis of the rectus.

Here is a woman who has had considerable tension on her sutures from the beginning and, if she vomited, she would have greater tension. For many years I did not use a supporting stitch, using ordinary catgut stitches, but one of the surgeons in this city had a case where the catgut stitch loosened during an attack of vomiting, and the intestines escaped into the subcutaneous fatty tissue and outside of the aponeurosis of the rectus.

In reopening the abdomen for one thing or another in these many years I have been impressed by the fact how little support stitches give after the second or third day. If you open the abdomen of a patient the second or third day after you have closed the abdominal wall, you will be surprised at the little support that stitches give. This led me to adopt this plan of using a figure-of-8 stitch, and I have never regretted it. It has, in addition to the advantage of a mechanical support up to the period of necrosis, the advantage of obliterating the dead space that would exist between the skin and the aponeurosis of the rectus. Obliterating this dead space as we do, you can see when we put on the gauze and subiodid of bismuth we have practically apposition of all tissues. You have, therefore, a primary agglutination, and, following the primary agglutination, you have primary adhesion of the tissues through the entire range of the wound. That gives us the nicest type of union and gives us all of the conditions for a good result.

In operating on fat people of this kind there is danger of colon bacilli escaping from the open intestine or open stomach into the fatty tissue. Colon bacilli will not infect peritoneum, as a rule, but a very small number of colon bacilli contacting with fatty tissue of this type will produce a suppuration, with a low temperature, the formation of pus, the rather rapid destruction of the fatty tissues, and a large pocket outside of the aponeurosis of the rectus.

I have been asked whether or not there is any trouble in removing the figure-of-8 stitches. No; I will tell you why. This is tied firmly on that gauze. There is on there now a continuous elastic pressure that cuts a large hole produced by the catgut in ten or twelve days, so there is no difficulty in removing it. Where it is a half-knot,—where the two cross below at a sharp angle,—if you are not careful in putting them in one will half-knot and allow of less pull, but not enough to be of any consequence or disturbance to the patient. It will give less disturbance than ordinary horsehair. We use a supporting stitch and the stitch does not cut the tissues crosswise or transversely as the tension on that finally would, differing from horsehair, which is elastic, and this prevents the stitch from cutting. Silkworm-gut is inelastic, like wire, and cuts transversely unless you have some protection, particularly here where you have considerable tension, and in this type of patients where you have a large, fat abdomen.

This wound is sealed with collodion, and a large dressing is applied and retained.

The final word has not been said—and, indeed, the middle word has not been said—with regard to the treatment of gastric and duodenal ulcers. We are not going to continue treating gastric and duodenal ulcers on the basis of gastro-enterostomy.

Just what we are going to do I do not know; but the reason we are not going to do that is this: Of the cases of duodenal ulcers that come to me, I should say that in one out of every ten there is a return of the symptoms after operation. This is also true in a larger percentage of cases of gastric ulcers, particularly ulcers for which we did a gastro-enterostomy in the last five years. In the last five years we did gastro-enterostomies and had frequent recurrence of symptoms. I said of ulcer—I would say rather in cases of symptomatic ulcer. You see, there is a type of ulcer you cannot find by palpation. In the gastric ulcers that do not produce a type of interference with the pylorus we believe that they do better without a gastro-enterostomy in the average case. It is the ulcer that is on the lesser curvature, close to the pylorus, or one which involves the pylorus, that calls for gastro-enterostomy. The ones higher up and those in front or back should be resected and can be resected, but when you look at this line [referring to chart] drawn through the stomach, showing the line of ulcer, you see that the enormous percentage—85 per cent. of the cases—of ulcers are in the zone to the left of that line, which interferes with the outline. The small round ulcer is not benefited by gastro-enterostomy, and we do not connect the stomach with the duodenum where we find these ulcers. We do not always find them. I recall the case of a young lady whom we had here in the hospital that was vomiting blood every day or two, and she kept it up for weeks and weeks, and we operated on her. She had typical hematemesis again and again, with a hyperacidity, with all the conditions that we get with ulcer, but no ulcer was palpable. We made a most careful examination. We did not open the stomach; there was no reason for this, and we did not do it. But, as I have said, no ulcer was palpable. We did not do a gastro-enterostomy. Finally, after putting her on the Leube treatment and keeping her for months on it the ulcer began to heal and she got perfectly well, and stayed well. But it was a long, tedious process, with the hemorrhage annoying us very much. We believe that if we had done the other thing in that case we would have had better results. On the whole, the immediate effects of gastro-enterostomy are satisfactory, with only about 1 per cent. mortality, and usually that mortality is from side issues, such as death from pneumonia or some intercurrent condition associated with the operation, with hemorrhage, or something of that kind. Rarely now is death due to defect in technic, because the principles involved in the technic of gastro-enterostomy are so well understood.

We were in England a year and a half ago, and, while there, saw as many as 30 surgeons doing gastro-enterostomies, and no two surgeons put in the same type of stitch, and yet they were all very well pleased with their results. What does this mean? It means that the stitch was an unimportant matter in the final results; but they understood the essential principle of the accurate approximation of serosa to serosa, with a thorough covering of the edges, and with an effort at obliteration of the sources for hemorrhage by carrying a top stitch on the edge. Those were the important elements they were governed by, no matter what the type of stitch was.

In this country we have a number of varieties of stitches and a number of varieties of technic, and we are getting about the same primary results that they are obtaining on the other side of the Atlantic. Take the men who are doing this special line of surgical work—like the Mayos, Moynihan, and Mayo-Robson—they are all getting uniform results, and no two of them do it in the same way. No two favor traction of the jejunum in the same way. We think it should be made in a definite way. It is our belief that the line of pressure in the intestine should be in the same axis, in the same general direction. We know, furthermore, that after a gastro-enterostomy is made the material will pass by the gastro-enterostomy opening and roll around and around in the stomach, as has been shown by the experiments of Codman and a Boston man, whose name I cannot at present recall. They showed that by giving bismuth meal it went around and around and passed the gastro-intestinal opening until it was properly prepared, and it did not stay, until nature had figured it out This shows the wonderful resources of nature.

HYDROPS

HISTORY

Male; age, twenty-five; occupation, laborer. Admitted December 21, 1911.

Family history negative. No tuberculosis or malignancy. Past history: Always well until four years ago, when he had a severe chill and felt feverish; he had a severe pain in his left side at that time; he recovered from this trouble and had no more sickness until the present trouble. Drinks tea and coffee, and takes an occasional glass of beer and whisky; venereal history negative.

Present Trouble.—Started on December 1st with an attack of some pain in the left side of chest, in the axillary line, increased on breathing; the pain was not accompanied by a chill or fever. He coughed at that time; the sputum was blood tinged; the pain in the chest has continued to the present time, as has also the cough.

COMMENTS AND OPERATION

Dr. Murphy: He had a chill with this attack?

Interne: Yes.

Dr. Murphy: Any blood expectoration?

Interne: Yes.

Dr. Murphy: What was the diagnosis?

Interne: Pleurisy. That is what the doctor who sent the case called it.

Dr. Murphy: What is the matter with the patient?

Interne: I do not know.

Dr. Murphy: Read the history of the present trouble.

[History of present trouble was read by the interne.]

Interne: He gives a history of pleurisy four years ago. He had an attack of severe pain in the left side of the chest in the axillary line, which was increased on breathing.

Dr. Murphy: Did he cough?

Interne: He started coughing at the time.

Dr. Murphy: Did he cough four years ago when he had pleurisy?

Interne: I do not know.

Dr. Murphy: What were the conditions that led to pleurisy four years ago? How long did the pleurisy last at that time?

Interne: I do not know.

Dr. Murphy: Has he had any cough between the attack of pleurisy four years ago and the present trouble?

Interne: The record does not state anything regarding that.

Dr. Murphy: Did he have malaise and a tired feeling, and did he have kidney manifestations for three or four, six or ten, or twenty weeks preceding the sudden attack of pain? Is this tuberculous pleurisy which this man has now, or is it a pure straight pneumococcus pleurisy?

Interne: The history states that he passed blood at the time.

Dr. Murphy: At what time?

Interne: December 1st; when the attack came on—when he had pain.

Dr. Murphy: How long did the pain precede the chill and fever?

Interne: Very shortly.

Dr. Murphy: That is very indefinite and is of no value. You must get at the facts in connection with the history of this case. While the patient speaks Italian, there are enough employes in this hospital who speak that language to enable you to get a more detailed history of this case, and this you should do. You should determine what is the cause of that pleurisy.

Interne: He says he began to have bloody expectoration when he got his pleurisy.

Dr. Murphy: Did he have bloody expectoration the day he got the pain? Is the bloody expectoration due to a pneumococcus infection? Did he have a circumscribed pneumonia, and is this effusion the sequence of pneumonia or a tuberculous infection in his lung? Is this pleurisy the same manifestation of an approximating infection of the serosa that we have when we have an effusion in the peritoneal cavity, or when we have tubercle bacilli in the tube, or when we have an effusion in the tunica vaginalis, or when we have tuberculosis of the epididymis, or when we have an effusion in the knee, or when we have tuberculosis in the upper end of the tibia? These are the questions you have to ask.

You have always to go back to the etiologic factors in making a diagnosis. How long has this man been in the hospital?

INTERNE: He came in Thursday, December 21st.

DR. MURPHY: What have you done to clear up the diagnosis? What are the findings?

INTERNE: The blood-count is 5000.

DR. MURPHY: That is of no special value except in the presence of an acute pneumonia. What are the findings in his sputum?

INTERNE: We have not a report on it.

DR. MURPHY: You ought to have a report. It is very essential to determine the cause of this man's pleurisy. We are told that this patient began to have bloody expectoration when he got his pleurisy; that he expectorates a blood-tinged sputum. Does a case of acute lobar pneumonia continue to expectorate blood-tinged sputum for twenty-three days? No; not at all. It never does so. He may have a necrosis or gangrene of the lung, and, as the result of the gangrene, he may expectorate an offensive material which will almost drive one out of the room, but not ordinary blood-tinged sputum. What is the temperature?

INTERNE: The temperature today is 97.8° F.; 98° F. in the afternoon at three. The highest temperature he had was 98.6° F.

DR. MURPHY: Is the sputum purulent?

INTERNE: It is not purulent. It is a sort of mixed.

DR. MURPHY: Why has he not elevation of temperature? Because he has not a mixed infection which always does the mischief with every tuberculous process. Remember, in tuberculosis in any part of the body, life is not materially hazarded by the tuberculosis except in the non-expansive zone of the cerebrum. *It is the mixed infection* with tuberculosis that destroys tissue and that kills the patient.

Let us go back to the tuberculous process and its association with pleurisy. In the first place, you have tubercle bacilli deposited in the lung weeks and months before the patient has a cough. Second, the patient has no cough to speak of except the dry, non-productive type until he has a mixed infection. Third, you have practically no elevation of temperature until there is a mixed infection. Fourth, the patient does not emaciate and sink rapidly until you have mixed infection.

In our analysis of an enormous number of these cases in general practice, accompanied later with a surgical practice along this line, these patients, like this man, begin to have a tired feeling in June, July, or August, but he was admitted in the winter with a feeble resistance. He had been exposed to cold. What does this mean? It means mixed infection. He began to cough in December, January, February, or March, and the cough stayed with him, and you [referring to the interne] charge his infection began with his cough which started in the winter, while the tuberculous infection took place in the dusty season of the summer. He doubtless got a secondary infection with his cold, and it continued because a tuberculous joint, when once infected, continues to discharge. It is the same proposition as tuberculosis in every part of the body. This history must be rewritten. It is incomplete.

I will tell you about the conditions that led to the other pleurisy and to the conditions that led to this attack of pleurisy. In our interpretation of this case that presents itself today, with no other manifestation of infection in any other position in the body, the question arises, is this a tuberculosis without a mixed infection? The effusion in his pleura may be due to the fact that he has a tubercular focus close to the serous lining of his lung, and that the effusion is the same type of effusion as that which goes with a tuberculous focus that approximates any serous surface.

You will recall that ten or fourteen years ago we aspirated these cases of pleurisy and empyema associated with tuberculosis, but we consider this now a waste of time. Why? Because the tubercle bacilli are not in the pleura at that time. The tubercle bacilli never go into the pleura. You rarely have a tuberculous pleurisy except where there is a rupture of the tuberculous focus into the pleural cavity, which is very rare. You have a pleurisy and an empyema associated with tuberculosis of the lung just the same as you have synovial fluid associated with tuberculosis in a bone close to a joint. You should not expect to find tubercle bacilli in the fluid, because they are not there.

This patient has had this effusion before, and he has it again. With this clinical history, what do we want to do? We want to favor the absorption of this fluid at the earliest possible date, because we know that the tendency in every position in the body in which a tuberculous focus is present is to have this tuberculous focus take on the character of a hematogenous infection with other microörganisms. You get it in the psoas abscess. You get it in a bone near a joint, and you get the mixed infections in the epididymis. Then comes rapid destruction. Here, we want to stop the flowing out of fluid and increase the absorption of it before we have a mixed infection occurring, because then we will have an empyema to deal with, and then we have the same difficult proposition that comes in the neighborhood of mixed infections in tuberculosis in every part of the body.

We believe this is not a pneumococcus pleurisy. In a pneumococcus pleurisy, as a rule, when there is much effusion, the fluid becomes purulent early. Why? Because pneumococci penetrate the lymph-spaces much more rapidly than tubercle bacilli, and then you have a pneumococcus infection with staphylococcus associated. In these cases, therefore, we may have an empyema following pneumonia, which is so common in children, and which causes one of the great misleading signs and leads to the omission or neglect of recognition of this condition in children, differing, too, from the condition in adults.

Let me give the symptoms of empyema in children. A child has a pneumonia and it does not clear up. When you use the stethoscope eight or ten or fifteen days after the pneumonia and you still have bronchial breathing, mainly tubular, right into the end of the stethoscope, you conclude that you have a case of failure of resolution of the lung in the child. Failure of resolution of the lung in a child is an extremely rare condition, and a negligible thing in making a differential diagnosis, differing in this from what you find in adults. In the adult, when you have an effusion, you have a distant bronchial breathing. It is away off in China, so to speak. It is not in the end of the stethoscope, and that is why empyema following a pneumonia in a child is often overlooked for weeks and months until it ruptures through a bronchus, and the child expectorates a large quantity of blood or pus and blood combined.

I cannot do better than illustrate what I have said by telling a story of my former partner, Dr. Lee, in connection with this matter. Dr. Norman Bridge was an analytic diagnostician. He did everything according to definite mathematic rules, and arrived at a diagnosis in a large percentage of the cases accurately. On the other hand, Lee displayed remarkable intuition in making diagnoses. He would just keep a few salient things in his mind, take the picture in at a glance, and arrive at a conclusion, and in this way he was a very able diagnostician. Dr. Bridge and Dr. Lee frequently met when I was a youngster, and I would hear them discussing these cases. Lee would say, "This is so and so. Can't you see that is what it is?" Dr. Bridge would bring his analytic faculty to bear on the case, and Lee would say, "Never mind your analysis," and it was remarkable with what uniformity and success Lee would make diagnoses. When he made a mistake, he would fall clear from the fifth story into the basement. But he would get the practical side right in a large percentage of the cases.

A practitioner from the upper Calumet region wrote to Dr. Bridge and said, "I have a case of pneumonia in a young child and it does not clear up." Dr. Bridge wrote to him and suggested that he had better puncture it, and he did. He wrote back that he had punctured it and had found a large quantity of pus. That told the story.

With a pneumonia in a child that does not clear up there is pus present, and it is pneumococcus pus with staphylococci, as a rule.

In adults it is a different thing. But, remember, the thing that misleads you is the bronchial breathing, and it is a clinical fact that tubular breathing is in the stethoscope in the child, and not so in the adult. Every pneumonia in a child that does not clear up in ten or twelve days should be punctured, and the physician will be rewarded with finding a creamy, yellow fluid.

In the management of these cases we have not resected a rib in any of the cases except where there is a communication with a bronchus. In connection with tuberculosis, you have first, as a rule, a serous and not purulent fluid. When there is a mixed infection you have purulent fluid, a thickening of the exudate. You have empyema with mixed infection with tuberculosis and the other bacilli, and then the patient gets hectic, and goes along with daily elevation and fall in temperature until he becomes cachectic.

In these cases you treat them, how? Not by resection of a rib unless you get the case in the very earliest days, and then it is unnecessary. In an empyema you have the same proposition to deal with, except on a more magnified scale, that you have in interosseous abscess or abscess in a large bone. Why does not an abscess in a large bone heal? Because the walls cannot collapse and come together. Why does not an abscess in the pleural cavity heal? Because the wall is not elastic and cannot collapse.

We go back to the method of treatment. Here is a case [referring to a diagrammatic picture], for instance, with an empyema cavity communicating with an artery rather than a bronchus. What the artist in this case intended to represent was a bronchus. We have a large abscess with a thickened wall, due to the reaction produced by the inflammation with the diaphragm holding it down, and with a thickened wall across the inside. Now, it is no trouble at all to open the chest. It is a simple thing to do this, but to close it after you open it is more difficult. Any one can open a chest. It is like asking a question: but to close it after you have opened it is a different proposition.

You take one of these old empyemas, and you have a thickened parietal peritoneum, a thickening of the visceral peritoneum and of the diaphragmatic peritoneum, and you have an inelastic, non-collapsible wall. Once you open it, you cannot close it. Then you have to do a Schede operation or a decortication and a Schede too, and still it leaks and leaks and leaks. Now, in working out this proposition—and this was one of our early uses of formalin and glycerin—how can we neutralize the material in the connective tissue in this case? The capsule of that is the connective tissue, granulation tissue, and the pus is a large, thickened mass, sometimes half an inch or more in thickness, and sometimes even shaped. The ribs become one continuous bony mass from one point of infection to the other—non-collapsible entirely. If you cannot cause that to collapse and heal, can you render the contents sterile? Yes; you can do this with great certainty. In our original cases—it is about seven or eight years since we began to do this—we started in with the injection of 10 or 15 c.c. of 2 per cent. formalin in glycerin. We inject a much larger quantity in the purulent cases, only two or three ounces after aspiration, and in a week we aspirate and inject again, and after a few days have elapsed we get the same quantity; that is, we aspirate and inject, wait a week, and then aspirate and inject again. We do the same thing until we get practically clear fluid, and then stop giving the injections. If you aspirate at the end of another two or three weeks, you will have straw-colored fluid with blood coloring-matter in it, and if you aspirate three or four weeks later, you will have plain straw-colored fluid. You have ordinary hydrops of the chest. The patient's fever disappears; his appetite returns, and he is building up, and you have merely a water-sac which is not hurting the patient in the least, but you cannot open that water-sac because the walls will not collapse. It must take time for the absorption of the cicatricial wall on the visceral side. Finally, as the adhesion and cicatricial tissue absorb, the lung gradually comes back and the chest gradually sinks in; the diaphragm comes up, the fluid is absorbed, and in a year and a half you find there is no fluid.

We have had a number of practitioners using this method since it was introduced in the treatment of this class of cases. One of these was the late Dr. Janeway, of New York. A hard thing to get men to appreciate is that you must expect something to be in that cavity until time has elapsed for the absorption of the connective tissue and the collapsing of the walls that surround it. When they percuss it, they say it is not any better. It is filled, however, with non-infectious fluid instead of being filled with infective fluid.

In the purulent cases we give them injections in that way. We render the fluid sterile and nature does the rest. If the patient should show a temperature after a puncture made by a sterile needle, it is evident that the necessary precautions were not taken. If it is clear fluid, let the patient alone. He goes on and gets well.

Any one who has tried to close a bad case of empyema after opening it and putting a drainage-tube in from one side to the other knows how important it is not to open it. I am sure if you have done this, that one experience will last you a lifetime, because we have all had our difficulties, and we have an easier way out of it. The fluid in this case is serous. We want to hasten its absorption and bring about agglutination of the lung to the side, obliterate that space, and stop the likelihood of purulent fluid coming out, because we believe this is tuberculous.

HEMANGIOMA OF THE LEG

HISTORY

Female; age, seventeen, admitted December 19, 1911.

Patient is a school-girl, aged seventeen; whose left lower limb shows the presence of numerous dilated veins.

Family history negative. Past history, has never been ill. Menstrual, regular twenty-eight-day type. Three to five days. No dysmenorrhea. Venereal negative.

Present Trouble.—Started when the patient was two months old; her mother then first noticed the dilated veins. Since then they have increased in size, and are more dilated when the patient walks, and cause a dull, aching pain through the limb while she remains on her feet. They cause no pain when the patient is recumbent.

COMMENTS AND OPERATION

This is an interesting condition. We have been on this case for some time, and we have done quite a little operating. It is a case of telangiectasis of the veins, with absorption of everything but the bones. There is nothing left but skeleton and skin. I can almost make my finger and thumb meet between the two metatarsal bones, because the absorption caused by the hydrostatic pressure of these veins has obliterated and produced absorption of almost everything except connective tissue. These are unpleasant cases to handle.

We commence above and we go down. We commence in the upper position, and ligate in the popliteal space. This morning we are coming into a bed of veins, which is one great varix. It will be ligated at this position again to produce obliteration and stop the oozing in this case. This case will probably terminate fatally in the end, even with amputation, and that is why we do not amputate. When these occur in the upper extremity, they are more dangerous than in the lower. When in the face, they are still more dangerous than in either of the extremities. I want to get just above the distention of the veins. This is a superficial vein which you see standing out. It is not especially significant. The deeper one, the one that is below the aponeurosis of the tibialis anticus, is the one that gives the trouble. We are right onto it, and I am going a little higher with my dissection to see if I can get to the aponeurosis of the muscle before I get into the vein. When you get into a real varix, you think of that hymn, "There is a fountain filled with blood," and here you are right at it, and it involves the periosteal vessels, which makes it so unpleasant to handle. This ligature will cut this muscle almost completely in two. While it is doing that, I want to try to produce a thrombosis in the veins; it will not permanently paralyze the muscle; it will only temporarily do so, because the ends of the muscle will unite again, and the muscle will become just as strong as it was before.

Now, we have tied this bunch of veins. In that varix the only pressure is venous. The venous pressure is scarcely a plus pressure. In the upper part it is a minus pressure during inspiration. Here it is only just a plus pressure. We reunite the aponeurosis, which will prevent any possible hemorrhage. The vein is opened on the side of the periosteum. I cannot close it, but I have provided and cared for that by being able to reunite this aponeurosis.

This is a congenital condition, and it starts as such and spreads and then often remains quiescent for years; then it receives the slightest trauma, and as a result of that trauma it starts to grow. Occasionally following a trauma it takes on the character that a nevus takes on by becoming wickedly malignant. When a nevus is present in any part of the body, and particularly nevi on the lower extremity, and becomes traumatized, it is prone to produce a venous sarcoma, or an angiosarcoma of a very wicked type. It is not, however, so prone to sarcomatous degeneration as is the pigmented dermoid. Where the pigment extends below the rete mucosum, you have the greatest danger following trauma. That produces a type of sarcoma which is the most virulent of all types of sarcoma with which we have to deal.

A peculiar thing about the sarcomata occurring in pigmented moles is that you have two types of malignant disease coming from them: First, you have the carcinomatous type, in which the epithelial cells are the active destroying cells. Second, you have the sarcomatous type, in which the mesoblastic cells or connective-tissue cells play the primary important rôle, and in both of these you have about an equal degree of malignancy. The tendency is toward a fatal termination.

A sarcoma that occurs in the pigmented mole in the lower extremity is transmitted through the lymphatic system. Sarcoma, as a rule, is transmitted from its primary position to its secondary metastatic foci through the circulation and not through the lymphatics. You have one other place in which there is a striking contrast to the law, and that is in sarcoma of the testicle, and particularly in sarcoma of the left testicle. Sarcoma of the left testicle transmits its metastases through the lymphatics, and you get secondary manifestation either at the bifurcation of the common iliac vessel, or you get it on the renal vein, where sarcoma often takes place. You know the lymphatics, as a rule, run along with the veins, and you have metastatic sarcoma occurring as a retroperitoneal glandular sarcoma following sarcoma of the testicle. When a sarcoma occurs in a pigmented mole on the lower extremity, you have a metastatic deposit or a pigmented sarcoma of the inguinal glands around the saphenous opening. So here you have a metastatic neoplasm occurring early; not only this, it seems to develop rapidly. Once these sarcomata become malignant, they spread from their primary position in the neighboring glands to the central system, occurring as a metastatic sarcoma in the lung or in some other portion of the body, having no definite point of election, but staying more frequently in the lung than the average sarcoma does.

Sarcoma in certain glandular structures has elective places for its manifestation or metastases. Sarcoma of the breast has its elective places, as well as carcinoma of the breast, or metastases in the upper ends of the long bones, as the femur and the tibia and the bodies of the vertebræ, and more particularly the latter. You have more metastases in these places from cancer of the breast to the bodies of the vertebræ than of any other bone; the head of the femur second, and then the upper ends of the long bones following that. Just why this occurs we do not know.

Again, we have sarcomata and carcinomata occurring in the thyroid gland, having definite places for election, namely, the shafts of long bones. There is a definite physiologic relation, however, between the election of the thyroid gland and the shafts of the long bones, because the thyroid gland controls the physiologic growth of long bones to a very great extent.

You will doubtless remember the experiments that were made by von Eiselsberg years ago, in which he took a litter of pups, removed all their thyroid glands, and after a definite period of time began to feed them each successively sheep's thyroid, and he grew litters of dogs of varying sizes and heights. He found by experiment that he could grow a pup to the size of a dog and a half or two dogs long, depending upon how soon he started in to feed the pups the sheep's thyroid. There, you see, is a definite relationship established between the thyroid gland and the growth of long bones. There is a definite reason why neoplasms in the thyroid and metastases in the long bones hold such a physiologic relation. But we know of no physiologic relation between the mammary gland and the short bones and the heads of long bones.

Again, in the metastases that occur you always have, of course, the same type of cells, whether it is a melanotic cell of mesoblastic origin or of ectodermic origin. That brings out the peculiar relationship between infections and neoplasms. There is nothing in any of the known infections that corresponds to that—nothing. If you have a tuberculous infection, if you have a gonococcus or staphylococcus infection in the foot, and you get a secondary deposit in the lung, the cells that play the rôle in the secondary inflammatory area and destruction are the cells of the lung, not the cells where the original infection was. Is this clear? If you have, for instance, a Neisserian infection in the urethra and get metastatic infection in the bone, you never have in the urethra the epithelial cells carried with that infection, and they do not reproduce in the new position. This is why there is one important dividing line between malignant neoplasms and all other types of infection.

The question arises whether this cell that plays the important rôle is a protozoan cell, or whether it is a stimulation produced in that cell by a living microörganism which tends to the multiplication of that cell—tends by this multiplication and by the toxic effect of the microörganism to digest the tissues in the neighborhood, and permit the epithelial or mesoblastic cell to go through the other structures, just as trypsin does in the leukocyte. Whether it is the cell itself that is a protozoan body, or whether it is something that is living in the cell that gives it that property, must be determined by the future, for it is not known now. But the method of its action is the method of action of a cell carrying a ferment which tends, first, to a multiplication of the cell itself, and tends, in the second place, to the absorption and destruction of the neighboring tissues, but always reproducing the cell itself of exactly the same type. Remember, too, when they are black pigment cells, you have a metastatic growth, always a black growth, too, carrying the pigment; not altering the nature of the cell in the least by the fact that it has been transmitted into a new environment; not altering it by the fact that its circulation has been increased and diminished both in transit and again in localizing itself on the new surface.

FISTULA IN ANO

HISTORY

Patient is a female, aged twenty-four, who enters the hospital on account of a rectal fistula. Admitted December 20, 1911. Family history negative. Has never been ill before present trouble. Uses tea and coffee in moderation. Occasional glass of beer and wine. Has never been married. Menstruation normal, three to five days. Regular twenty-eight-day type. No dysmenorrhea. Venereal negative.

Present Trouble.—Patient had an abscess just adjacent to the external sphincter of the rectum in October, 1908. This abscess was lanced and discharged the pus from the outside just adjacent to the sphincter. This abscess then healed up in one week and caused no more trouble until August, 1909, when the site of the old abscess commenced discharging a few drops of pus every day and became slightly tender. This tenderness and discharge lasted up to the twenty-fourth of November, when it was operated; the operation consisted of suturing the proximal opening of the fistula and approximating the margins of the opening with sutures. The external fistula opening was injected with Beck's paste at the time of the operation and also one week subsequent to the operation. Two weeks later, on December 8, 1911, the discharge recurred and the conditions were absolutely the same as they had been previous to the operation.

COMMENTS AND OPERATION

The practitioner who operated on this patient inserted his sutures in an infected field, and what should happen? Just one thing should happen, namely, failure of union. What did happen? A failure of union. Then, in addition to that, he plugged up the fistulous opening from behind. This meant an accumulation of feces and gas, with a permanent channel established between the bowel and the surface around the plug of Beck's paste. Beck's paste is not intended for this purpose. The paste has a definite place in fistula. It acts as a plug here.

In the management of these cases of fistulæ we must divide them into, first, the fistula that occurs on the anal side or the mucous surface side of the sphincter; second, the fistula that comes through the sphincter, taking in a considerable portion of it, which is rare; and, third, the type of fistula that passes from the mucosa above the sphincter out into the anus; fourth, the dissecting type that enters high up, two, three, or four inches, and dissects all around the rectum, making various channels, some of them opening up under the symphysis, others opening on the one or the other side of the navel. These must be differentiated before you do a single thing in an operative way. If a fistula is properly treated in an operative way, you may effect a permanent cure, or if it is improperly treated, the patient may have permanent incontinence of feces.

I have a very vivid recollection of a man who came into my office with a fistula from a previous operation. He was a traveling man. He had been operated upon by a specialist in this branch of surgery, and as I asked him to remove his trousers he took out a gun, laid it on the desk, and when I asked him what he had it for, he said, "I am waiting to use that the first time I can find that fellow." Why? Because this man had to wear a napkin and was doomed to wear it all his life. He was doomed to wear a napkin because the man who operated upon him did not make a differential diagnosis, before he operated, of the fistula that went above the sphincter and the one that went below, and he divided the sphincter twice, contraction occurred, leaving a permanent canal for leakage. This was an unpardonable thing. I ask you to put yourself in that patient's position. If I were the patient, my love and admiration for the surgeon who did that operation would not be very great.

These errors are avoidable if the proper precautions are taken. The surgeon failed to differentiate between the various fistulæ. This man was operated on the outside and operated on the inside.

The first important thing for us to determine is, where does this enter the bowel? If we merely operate on the outside without finding the entrance of the fistula to the bowel, we would have a return of the condition. If we operate on the inside without finding the entrance, we will have a return of the lesion. In this case we are going to endeavor to feel our way through by putting this probe through different grooves in order to get the inlet to the bowel and see where the lesion is. I find a depression here on the inner side, and whether that depression communicates with the fistula or not I do not know. I feel the probe now, which is not very far from my finger, but it is not in the bowel. I will stretch the bowel a bit. Here appears to be the point of non-union. There is a resisting cicatricial mass right here. There is the probe now, down near the bowel, but it is not in the bowel. It is outside of the mucosa, and decidedly so. It passes well around the side without any force on my part. It is now close to the surface of the mucosa, but is not in the mucosa. The opening should be high up from the position of the probe. There seems to be a burrowing ulcer close to the mucosa of the bowel, and somewhere in that neighborhood, but it does not penetrate it, that is, the instrument does not go through. Each time I come back to the cicatricial lump. I will see if I can, by inspection, find the opening.

Here is an opening, you see, that goes into the perirectal space. Now then, we expect these two openings to meet. They do meet. This is all you see. There is the sphincter behind. It is all on the lower side of the sphincter, and anything I cut is going to be outside of the sphincter. There you see the connective tissue, and I am following it down. Notice the Beck's paste coming out of the fistulous opening. There is the internal opening. Here, you see, is the packing, and here is the tract. There is the cicatricial tissue of the tract, and I am dissecting out this fibrous tract. Here I show you the true sphincter, and there is the canal right through. Let us see if there is any more bismuth paste forced into the channels in the neighborhood. We have all the pockets. Here is the sphincter. See it! It is inside the tract. It does not interfere with the field in which I am working. I am not taking up the mucosa at all. There is the connective-tissue margin. The connective tissue, you see, completely encompasses this tract, or the place where this tract was removed. It gives an exit entirely inside of that and no exit outside of the sutures.

We shall fill this with subiodid of bismuth, keep it open, and healing will take place more rapidly.

ARTHRITIS OF THE WRIST-JOINT

HISTORY

The patient is a chauffeur, aged twenty-nine, who was admitted to the hospital because of an arthritis of the left wrist-joint. Admitted December 22, 1911. Family history negative. Has never been ill before the present trouble. Had gonorrhea two years ago, January, 1909, and again contracted the disease on the twenty-fifth of October. Still has a discharge. Uses tea and coffee in moderation. An occasional glass of beer. Bachelor. Denies luetic infection. Neisserian infection twice.

Present Trouble.—Started on December 6th. The right elbow became painful, but did not become swollen or hyperemic; the pain lasted twenty-four hours and has not returned. On December 12th, six days later, the left knee pained and became slightly stiffened; this lasted five days. On December 12th the left wrist became very much swollen, hyperemic, and painful; the pain was dull and throbbing. The redness and swelling have gradually subsided until at present the wrist is slightly swollen but not hyperemic. The pain has entirely disappeared, but the wrist is stiff and the patient is unable to use it.

COMMENTS

This is a case of wrist-joint infection. The patient is in the third week of his Neisserian urethral inoculation. He has aches all over his bones, followed by general muscular rheumatism involving the right wrist-joint. This is the right time to save the wrist-joint. He has metastatic infection in his wrist-joint from the urethral infection, and he does not come in, as these patients usually do, with deformity and their joints out of position, but he comes in before the serosa is destroyed. He will receive an injection of 5 c.c. of a 2 per cent. solution of formalin and glycerin, prepared twenty-four hours before it is used. He will have another injection in a week from today, and that is probably all he will have, and there is no doubt but that with this treatment he will have a perfectly movable and normal wrist-joint. Why? Because he is getting this treatment before the articular surfaces are destroyed or agglutinated.

VOLUME I *NUMBER 2*

THE
SURGICAL CLINICS
OF
JOHN B. MURPHY, M.D.
AT
MERCY HOSPITAL, CHICAGO

APRIL, 1912

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NOTE: On account of the haste involved in publishing the February number of the Clinics, a few errors of omission crept in, which will be avoided in future issues.

Volume I SURGICAL CLINICS OF JOHN B. MURPHY Number 2

UNUNITED FRACTURE OF THE TIBIA

TRANSPLANTATION OF BONE

HISTORY

PATIENT is a civil engineer, aged thirty-two, who was admitted December 27, 1911, because of a fibrous union following a fracture of the tibia and fibula of right leg at a point two-fifths between the malleoli and the patella.

Family history negative. Past history: Amebic dysentery in 1908, good recovery. Malaria for the past ten years. Contracted these in the Panama Zone. Takes an occasional glass of whisky. No tobacco. Absolutely denies venereal infection.

Present Trouble.—On June 9, 1911, the patient was in a railway wreck, and his tibia and fibula were crushed between two seats of the car; a comminuted compound fracture resulted; also a division of the anterior tibial artery at the site of the fracture. The point of projection of the bone was the site of a pus discharge which started on the third day and continued to discharge for fifteen days. At the termination of this period the external wound was entirely healed. The patient ran a temperature ranging from 100° to 102° F. for the first three days of the discharge. With the exception of the temperature, he had no other general symptoms. The limb was placed in a cast on the fifteenth day following the fracture. The skiagraph taken on the day following the putting on of the cast, June 24, 1911, showed an overriding of both tibia and fibula of 1/4 inch. The leg was angulated inward 45 degrees. No extension was put on. The cast was removed in four weeks, and examination suggested the presence of soft callus, with a very slight motion at the point of fracture. Another cast was placed on the limb and allowed to remain on for one month. The patient was allowed to walk with crutches six weeks following the accident. At the end of the second month, when the cast was again removed, the callus was a little more solid, but there was still a slight movement at the point of fracture. A plaster-of-Paris collar 12 inches wide was placed around the point of fracture, and allowed to remain on for three weeks, during which time the patient walked with crutches. The collar was removed at the end of this time, three months after the accident. The patient used crutches for two and one-half months; at the end of this time a second *x*-ray was taken, December 5, 1911, six months after the accident, showing an overriding of ½ inch, with considerable mobility at the line of fracture. This mobility has increased slightly and progressively up to the present time. A fragment of bone accounts for the apparent overriding of a great degree.

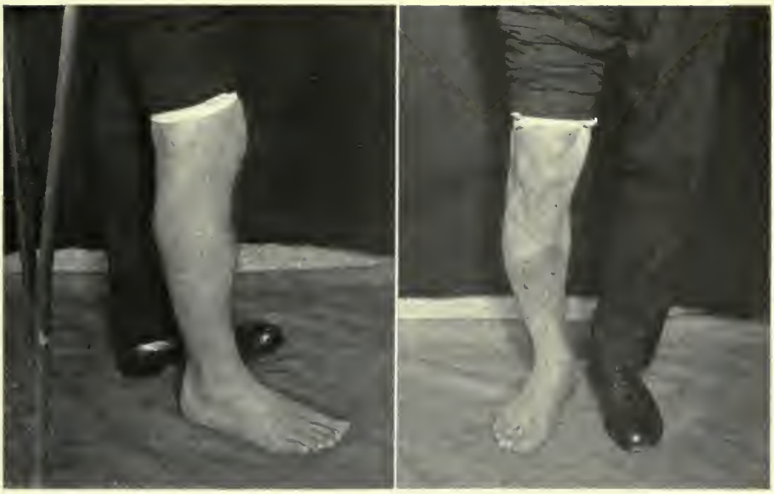
COMMENTS AND OPERATION

Dr. Murphy: What was the immediate preparation of the wound?

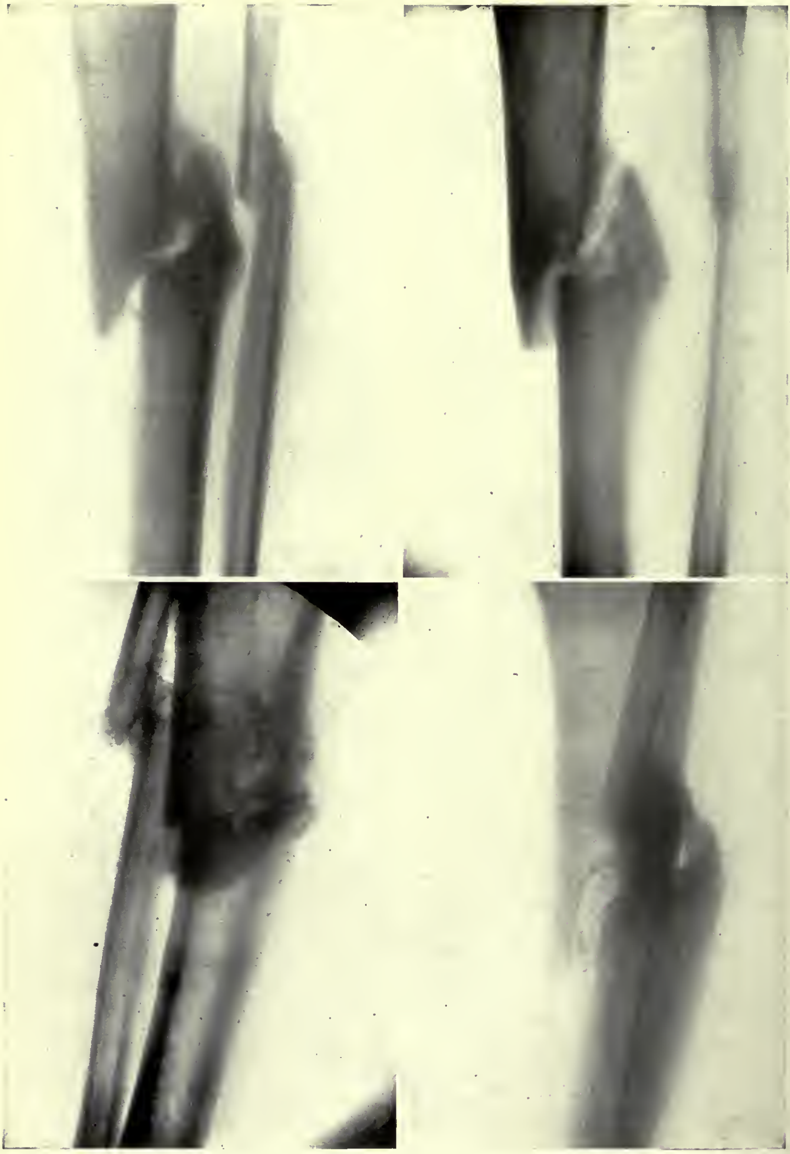
Interne: I do not know. The history does not say.

Dr. Murphy: That is a very important item for you to get and incorporate in the history, doctor, because we are trying to figure out why these compound fractures become infected in the primary dressing.

When I was in the Alexian Brothers' hospital we had this experience: whenever a case of compound fracture from indirect violence—as where a man fell and a bone penetrated the skin and came out—was brought directly to the hospital and had not been touched until he was brought to the hospital, where aseptic precautions were taken immediately, we found that these cases year in and year out went on and we got primary union. We never handled them, but simply freshened the surfaces, after removing the dirt, and they all did well. But whenever such cases were examined or handled by the police surgeon or by some one else at the time when the man was injured, or a doctor put his finger in to see how badly the bone was broken, the results, from the standpoint of primary union, were unsatisfactory. There was generally infection. When these patients were brought to the hospital immediately after the accident, without



Figs. 9 and 10.—Case of ununited fracture of the tibia before operation, showing deformity.



Figs. 11–14.—Skiagrams showing ununited fracture of the tibia. These four skiagrams were taken at different angles.

any disturbance of the wound, with nothing but a primary conservative dressing having been applied, we got primary bony union with great regularity. Do not let any one touch the inner surface of such a wound. Clean it out with scissors and forceps —never by washing or disinfectants of any kind. We learned early in our experience that disinfectants were injurious; that all solutions retarded repair and favored primary infection. We do not use solutions, as you have noticed, in the abdomen, *not even* salt solution in the peritoneal cavity.

You have heard the history of this case. The accident occurred on June 9th of this year (1911),—six months ago,—with failure of union, but with very good apposition of the bone, as it appears in the skiagram. We have four skiagrams, showing beautifully how misleading a single skiagram may be, and yet how thoroughly instructive it can be. One skiagram shows bony union, while another shows that there is no bony union. A third skiagram shows partial bony union, and a fourth shows there is partial failure of union. These skiagrams were taken at four different angles; they show the different phases as the tube was rotated at different positions. *This accentuates the importance of paying more attention to these things, and of being careful in drawing conclusions from the x-ray.*

The reason this man has no bony union is not because the bones are not close enough, but because the ends of his bones are not producing new bone. The osteogenetic force has ceased at some place in the process of repair. This picture shows that the fibula is united, but that the tibia is not. The greatest hazard in this whole case is necrosis of the flap over the implant following the operation.

We make our incision so as not to disturb the parts where the skin is thinnest. The groove for the implanted bone will be on the same plane with the divided skin. Of all places in the body where asepsis is required in a superlative degree to get results, it is here in working with these bones. There is a firm fibrous deposit between the ends of the bone, extending the entire *width* of the non-union. It is ¼ inch thick, interposed between the overlapping fragments of the bone, as shown in the skiagram.

I am now reaming out the medulla above and below the line of fracture. There is the interosseous splint which I just took out from above. I have taken out the medullary splint which nature had formed in both ends, but not across the line of union, as you see. In removing the transplant from the other tibia I am going just to the inner side of the tibialis anticus, and reserving the periosteum with the fragment. I thought at one time this was going to be easier done with the saw, but I have changed my mind about that. The chisel is the best instrument for this work, both as to time and direction. You see now, as I enlarge *the groove of division*, I enlarge it toward the compact bony tissue and not toward the flap. If you enlarge it toward the bone-flap, you are likely to split the bone-flap, which occurred in one of my cases. I now divide the crest of the tibia with a saw at both ends of the transplant, and with the chisel fracture it off. You see, I had mortised out a place for this and now have put it in its bed, and I am covering it first with a deep row of sutures with which I bring the soft parts and periosteum together. There is a large vein on the side of the field standing there all the time, and I have succeeded in avoiding it. The skin is closed with catgut suture.

In the development of non-union we take into consideration the several elements that go, first, to make up a union, and from these analyze the conditions that go to make up a union following a fracture. When you come to analyze the conditions that go to make up union, you ask yourselves the question, Which one of the elements failed in bringing about union?

I have taken all the tension off the line of skin suture that I possibly can. We will have quite an oozing from this wound for about forty-eight hours, and during that time blood will come out in considerable quantity. We provide for that by the carbolic dressing which we use, and which is the dressing of the preaseptic period or the antiseptic period, and is intended to care for this exudate when it comes to the surface. It absorbs it. We put on 5 per cent. carbolic gauze, which prevents the secretion from undergoing decomposition, which ordinary sterile gauze does not do. We put this limb in an ordinary straight splint, or a wire trough. The leg is strong enough already to go on doing business with that bone splint in there, but you immobilize it so as to get granulation coalescence *across the line of apposition.*

Here is a picture of a case which accentuates the point I made in connection with this case, namely, that while the bones appear to be almost in accurate position, there was not the slightest effort at union. Take the case in which the Lane plate was used. (See case of Ununited Fracture of the Humerus in this number.) There was splendid apposition, so far as approximation was concerned—just a slight lateral displacement which amounts to nothing, and still there was no effort at the reproduction of bone. In the second picture I show you you see the other side, the side on which the plate was inserted, and there is perfect alinement. We have a third picture, after we took off the plate and waited for the granulation tissue and the mild suppuration that was present to cease. This plate was used in a compound fracture, which never should have been done. We did not have to wait long in either of these cases, as there was no necrosis. We took out the foreign body and allowed the granulations to coalesce. Here are two pictures taken before the implantation of the spiculum of bone. The first showed considerable displacement, and the second a perfect alinement, the same as before, and still no effort at union.

These pictures, after operation on these patients, show the various stages of regeneration.

TREATMENT OF FRACTURES, SIMPLE AND COMPOUND

Regeneration of Bone

When we come to the modern treatment of fractures, we have these things to take into consideration: first, the treatment of a compound fracture; second, the treatment of a simple fracture. These are the two grand divisions. We will take up compound fracture first, because a compound fracture that is badly managed leads to more evil results than a simple fracture badly managed. In a simple fracture you can get faulty union or failure of union with deformity, but you have not materially hazarded the patient's limb or life at any part of the process. A compound fracture, on the other hand, is not only dangerous to the limb, but dangerous to life. Every compound fracture must be converted, practically, into a simple fracture, regardless of the deformity. You must get as good apposition as you can, which does not interfere with union of the soft parts.

In all compound fractures the first step is the primary preparation. You should never wash, scrub, handle, or touch the lacerated surfaces either of bone or soft parts. In preparing the surrounding field, which is the first thing that should be done, soak the entire field with the hair on with the ordinary tincture of iodin, or in children with a 50 per cent. tincture strength. If there is grease in the neighborhood, it is desirable to remove it with benzin without letting the benzin flow into the wound. When the neighborhood is disinfected, then the freshening of the surfaces of the wound is the next thing in order. That should be done with the scissors and forceps, and possibly with the aid of a chisel or bone-cutting forceps, to take off points of bone that are protruding. Do not put an instrument of any kind into the wound. If you have a sequestrum that is separated and loose, pick it out with an instrument; otherwise do nothing. Remember that a sequestrum in a compound fracture adds to the probability of infection in the wound and should be lifted out, but never inflict additional trauma, and if the periosteum is attached to the sequestrum, permit the sequestrum to stay. If it is free,—entirely detached,—then it is going to be a dead foreign body, no matter what you do, and it should be removed, because it lessens the likelihood of infection in the wound. Next freshen the surfaces of the skin and make them clean-cut where they are contused, so as to get an accurate apposition of the cut skin-edges with horsehair, which we prefer, and with here and there a tension suture of silkworm-gut, if necessary. Finally, dress the limb with a large dressing capable of absorbing the oozing from the ends of the bone and from the lacerated tissue, and maintain them sterile if the dressing takes up the secretions; the best of all preparations that we have had for that purpose is the 5 per cent. moist carbolic gauze. You understand, this is not a disinfectant: it is merely material to prevent decomposition. Where you have saprophytes on the skin in the immediate neighborhood, you have decomposition, and the carbolic gauze will prevent decomposition of the escaping fluid.

Immobilize the limb to the best degree, and dress the fractured portion in an elevated position to insure a better return circulation and prevent edema. Keep it immobilized and without disturbance, if possible, for ten, twelve, fifteen, or twenty days, unless the patient has a temperature. The indication for changing the dressing is not the primary elevation of temperature. You have two elevations of temperature—one primary, which was known as a fibrin temperature in the early days, or the breaking-down of the blood-clot, which occasionally causes elevation of temperature to 100° or 101° F., rarely higher. That subsides. That comes on within forty-eight hours and subsides within another forty-eight hours, and the temperature returns to normal, as noted in this case history. If the temperature continues in the second forty-eight hours up to 100° to 103° F., you must carefully separate the dressings without handling the limb, permitting the wound secretion to escape. It may be retained by a stitch. Open it, allow the wound secretion to escape, and put on again a large carbolic dressing. You never put in a primary drain. This dressing is not disturbed until you have final healing of the soft parts if it goes through an aseptic course—and it will in the vast majority of cases of indirect compound fractures.

In the crushing injuries of railroad accidents, and of cars passing over the limbs, and accidents of that kind, which usually require amputation, you have different conditions for infection, which you must accept and handle accordingly.

Having converted a compound fracture by this method into a simple fracture, you are ready for the process of repair of a simple fracture. Never wire a *compound* fracture. Never plate a *compound* fracture. Never put in a foreign body in a case of *compound* fracture. We have been taking them out for thirty odd years, and the number that have healed in, put in there primarily in compound fractures, in my observation in that great period of time can be counted on the fingers of one hand.

That is an experience, and it is a large experience, not of my own work, but the work of others, and my experience has been quite in accord with that of other surgeons, so that for a long time now we have entirely abandoned the insertion of a foreign body, for the best reason in the world, its failures. A wound immediately after a trauma has its lowest possible resistance to infection. Its vitality is lowered. Wait until it is restored, and then, if you have to plate a compound fracture or what has become a simple fracture at the end of ten days or two weeks or three weeks, you have granulation tissue formed, a regeneration of tissue established, with blocking of the lymphatics, and you have all the conditions for primary repair. There is one place where there is an exception to this condition, and that is in fractures of the jaws. There you can heal in wire and many other materials, as was beautifully illustrated by a case in which we used silver wire and have had it stay in indefinitely in the jaw. It heals in there on account of the enormous vascularity of the tissue involved and the great resistance which it has, and its ability to overcome infection of itself and around the foreign body.

Now we go back to the process of repair of a simple fracture that leads to union and the elements that are essential in the process of repair. In a simple fracture you have, first, hemorrhage, not alone between the ends of the bone, but into all the neighboring structures; that is, the trauma and foreign material —*i. e.*, extravasated blood—produce irritation of the nearest living cells, which immediately commence to multiply. They produce irritation of the nearest capillary networks, which immediately commence to send out their granulation loops into the blood-clot, finally remove it, and when the blood-clot is removed, you have a large mass of connective tissue where you formerly had a blood-clot. That connective tissue has a tendency always to contract. You have in the medulla and in the periosteum and in the intermediate space granulation tissue forming from the capillary loops of the Haversian canals. You commence, then, to have this vast mass filled with granulation tissue, which finally becomes fibrous tissue, and in every structure you have an effort at the reproduction of the normal tissue which contacts or approximates the distal end or portion that was lacerated. Each histologic structure endeavors to reproduce the same type of tissue that it had before. Finally, the result is the production of connective tissue uniting the ends. The arteries finally develop a collateral circulation, through buddings from their occluded ends. The nerve overcomes distance, if it is not great, with loose connective tissue, and later with the axons passing through; the bone forms a soft callus, and then it has osteogenetic forces, which are three: subperiosteal layer, the osteoblasts on the walls of the Haversian vessels in the compact bony tissue, and the blood-vessels or capillaries that bear the osteoblasts in the medulla. Those are the three sources of reproduction of bone. Those are the osteogenetic elements. Finally, the capillaries coalesce across the line, and then you have a contacting of soft tissue. Then comes ossification on the walls of the blood-vessels, forming the cylinders of bone around those blood-vessels,—lamellæ,—first, in the periosteum, second, in the medulla, and third, in the compact bony tissue, until you have these loops passing across and uniting. First, you have the external callus ossified; second, you have the internal callus, and third you have a definitive callus, when union is complete. That is the process of repair, in that, with the evolution of bone, you have the development of cartilage cells, which ossify. In the development of bone, when you make *autogenous* transplantation, as, for instance, in the bone I put in now, you never have a cartilaginous stage.

When you have non-union, what happens? When you have non-union, this process of repair can be arrested in any one of the stages that I have just mentioned. First, in a compound fracture you have external bleeding and absence of blood-clot to support the capillaries from above and below, and the primary scaffolding for the loose young connective tissue is gone; second, in the repair of that primary scaffolding you have the breaking-down of the blood-clot by infection, and the capillaries come out from both sides above and below in a suppurating zone, with the lymphocytes and connective-tissue cells being killed again and again, as they come into the gap by the toxic and biotic effects of the microörganisms that are producing the pus; so that you have column after column of soldiers that have come in from the neighboring connective tissue killed off, but they keep on surviving in the deeper layer of connective tissue until finally the connective tissue holds them up across the line and the capillaries coalesce, and you have union of the soft parts across the line of a compound fracture; from then on the processes of reproduction of bone are identical with those processes that go on in a simple fracture, that is, from the time a soft callus is formed.

The reason you have non-union very frequently in compound fractures is that when the connective tissue is forming in here to span the space between the ends of the bone you have fibrous tissue, fascia, and muscle from the side drawn in by the central contraction. Always remember that connective tissue has a tendency to contract. Drawn in by the central contraction you have a mass of fibrous tissue or capsule formed between the two bones. That is why you have more frequently failure of bony union in a compound fracture in the second stage of the process than you do in a simple fracture. On the same basis you can have this in a simple fracture and compound fracture both; you *can* have soft callus form, but it may not form. You may in a simple fracture have space between my ring and middle fingers filled with cystic blood-clot, surrounded by flat-cell endothelium, resembling endothelial cells of the synovial membrane and peritoneum, developed from the endothelial cells from the lymph-spaces around the blood-clot. As the solid blood elements are finally absorbed from them, you have fluid in the center; you have a cyst between the ends of the bone, preventing union. When these fragments of bone are brought together, you can have cartilage developed over the ends of the bone, and through friction finally a metaplasia of the connective-tissue cells, produces a membrane resembling synovial membrane, and lined with cells resembling endothelial cells, you have then a true false joint formed. You have a true pseudo-arthrosis. Those are the various stages. When you have the granulation tissue approximated, you can have an entire failure of effort on the part of the osteogenetic cells of the deeper layer of the periosteum, the osteogenetic cells on the wall of the Haversian vessels in the compact bony tissue, and of the osteogenetic cells in the medulla of the bone to reproduce bone. You can have failure of each and every one, or of any one of these, all making no effort at the production of bone-cells, and taking on calcareous matter. You have a fibrous arthrosis with the failure of the osteogenetic elements. Why that is no one has figured out. There have been many theories advanced. One theory is that it is a local condition; another theory is that it is a constitutional condition. From our standpoint, we believe it is a local condition, and not a constitutional one, in the majority of cases, because the fibula will often unite, and the bone right next to it, the tibia, will fail to reunite. The tibia will be broken in two places, and it will not unite; you freshen the surfaces, and still it will not unite. You resort finally to the Court of Appeal, which is the interposition of a bony splint. In the production of bone it was the fourth stage of union which failed in this case. There was a failure of effort to reproduce bone-cells. Here [showing *x*-ray] you can see the periosteum producing around this fragment a little bone, but in the rest there is no effort at all at reproduction of bone. I went through the upper surface of the bone and opened in there. I might have drilled a hole so that I could have put a splint across in that direction [indicating], and be able to reduce the deformity. But I made no effort to do so because that deformity has nothing to do with the bony union.

What rôle will the implanted bone play? The implanted bone, the compact bony tissue, always dies—whether it is large or small, it first dies. The rôle it plays, therefore, is merely a passive rôle. It is the rôle similar to that played by the blood-clot. It gives a scaffolding through which the Haversian vessels travel through the Haversian canals of the transplanted portion from one end to the other of the bone. They carry with them two cell arbiters or workers—one, the osteoblasts, and the other, the osteoclasts. The osteoclasts dissolve the compact bony tissue I have transplanted, and all that disappears. They produce osteoporosis. The osteoblasts are also carried along these Haversian vessels, and commence osteal production or regeneration, and you have finally a filling in of all that dead space with new bone which is identical in size and shape with the fragment that has been transplanted, as is illustrated by this case. You see the osteoporosis coming up there. There is the end of the bone [indicating]. The porotic lines come closer and closer, isolating and filling in until you have complete coalescence across the gap of the interposed portion. You have then new bone formed of the size of the interosseous splint, and, as indicated in this picture [illustrating], it grows to any size that is necessary for the support of the limb.

Then comes the final act in every bony union—the act of absorption of the excess of bone. You have, by the time this is united, a medullary splint, a subperiosteal splint, and an interosseous splint in a normal union. The process of absorption commences, and you have external and internal callus gradually removed, so that you have formed a new continued medullary space, and you only retain enough of bone to give necessary support. Nature removes the excess of bone that is there. If there is a fracture near a joint, then you have deformity, and nature throws in more deposits until you have a callus ½ or ¾ inch thick, trying to hold up the weight that is too great for her to carry.

In the transplanting of bone in this way you do more than you do by any other method of approximation—you put in a material scaffolding, which carries osteogenetic layers from one fragment to the other. If that piece of transplanted bone becomes loose or septic, the fragment will have to come out. We might get union as a result of that, but the fragment of bone will have to come out. It will be a dead and septic foreign material, just like a sequestrum, and it will have to be removed. The only graft you can use is the human graft, and that is best taken from the same individual. That point has been definitely settled. The other grafts are all digested, and the element necessary is that the bone which is to regenerate this graft must contact with osteogenetic living elements.

In some of my earlier work on transplantation of bone twelve years ago I transplanted a patient's finger into a bad saddle-nose. A patient came in to have a plastic operation done on the nose. Another patient came in at the same time with double thumb: I amputated one thumb, and, after removing the skin, transplanted it into the nose, and it healed in beautifully, but I had not learned an important lesson, *i. e.*, that transplanted bone must come in contact with freshened periosteal denuded living bone. I did not take off either of the periosteums so that they could contact. It lived. It healed in beautifully. I showed this case as a brilliant result of transplantation for the correction of a deformity, but as time advanced every portion of that transplanted bone disappeared. Why? Because it was not in contact with osteogenetic living elements. The periosteum of transplanted bone plays a secondary rôle in the regeneration of bone only, but that it does play a secondary rôle is beautifully illustrated in the work of Macewen, and in some of the recent experiments, where the periosteum was peeled off for two or three inches and left as a span, the *x*-ray showed the reproduction of bone. What a year or two years' further investigation will add to this nobody knows. Wherever we have transplanted bone with or without periosteum, if we did not contact it with osteogenetic elements, it finally disappeared by absorption.

I would like to do that case in which we had this failure over again, because I am now just as certain as two and two make four that I would get a satisfactory result.

In the case before us the only slip that might occur is sepsis, otherwise it will be mathematical in its results, because we are observing the definite and positive laws controlling the regeneration of bone which I have laid down.

CHARCOT ANKLE-JOINT

HISTORY

Male; age, thirty-five. By occupation a liveryman. Always lived in Ohio. Married twelve years ago. Two children, seven and five years of age, both living and well. Family history: Father died at age of sixty-seven from stomach trouble; mother living and well. Five children in family, all living and well. Past history: All diseases of childhood. Diphtheria twenty-five years ago—complete recovery; no paralysis. No typhoid, scarlet, or pneumonia. No jaundice, rheumatism, or tonsillitis. No operation or injuries except present trouble. Neisserian infection at about age of twenty—fifteen years ago. Says he had chronic urethral discharge for six months. Denies any chancres. Has had some secondary symptoms associated with his urethritis, such as sore throat, falling of hair, and adenitis. For past two or three years has had severe pains in his bones, especially in tibia, more at night than in day-time. Both pupils react very sluggishly to light. The Romberg test is markedly positive, although he complains of no trouble in dark. Patellar reflexes entirely absent. No gastric crisis, bladder or bowel symptoms.

Present Trouble.—Dates back to March 13, 1910. Patient states that at that time he fell from a hay-loft to the floor below, a distance of 18 to 20 feet, and struck on his right leg, on the inner side of foot. *It gave him no pain, but the ankle would not support his weight.* A doctor was called, who said he had fracture of right ankle, and said bone did not protrude through skin. Physician put a circular cast on it,—within thirty minutes after injury,—extending up to knee-joint and including entire foot. No pain after cast was put on. Cast was left on for seven weeks, during which time he was about on crutches, but did not touch the foot on the ground. At the end of seven weeks the cast was removed. At this time the ankle appeared to be in good position, he could move it without pain, and it was only slightly swollen. Five weeks following removal of the cast he was allowed to walk about on crutches and began to put a little weight on his foot. At the end of another five weeks he walked without crutches and attended to his duties. Patient says the conformation of the foot at this time was perfect. Was up and about on his feet nearly all day. About two weeks after this he began to notice that there was a little pain in the right ankle—only when he walked. Says he continued to walk about, but pain gradually increased, and he also noticed that it was a little swollen, and gradually the ankle was beginning to bow out a little. These symptoms increased until, at the end of five weeks, his right leg and foot up to the knee were swollen to twice their natural size, was bowed out at ankle-joint, and became so painful that he could not walk on it. He went to bed two weeks, and swelling and pain gradually decreased, but did not entirely disappear, and deformity at ankle remained. He had no chills, fever, nausea, or vomiting. For next seven months he got about on crutches and noticed very little change. At end of this time he began to walk without crutches, and has continued in this way to the present time. His foot is swollen at ankle-joint and deformed. He has a little pain in it when he puts his weight on it, but no pain when at rest. His general health has been good; no loss of weight. Appetite good. No night-sweats or cough. Bowels regular.

COMMENTS AND OPERATION

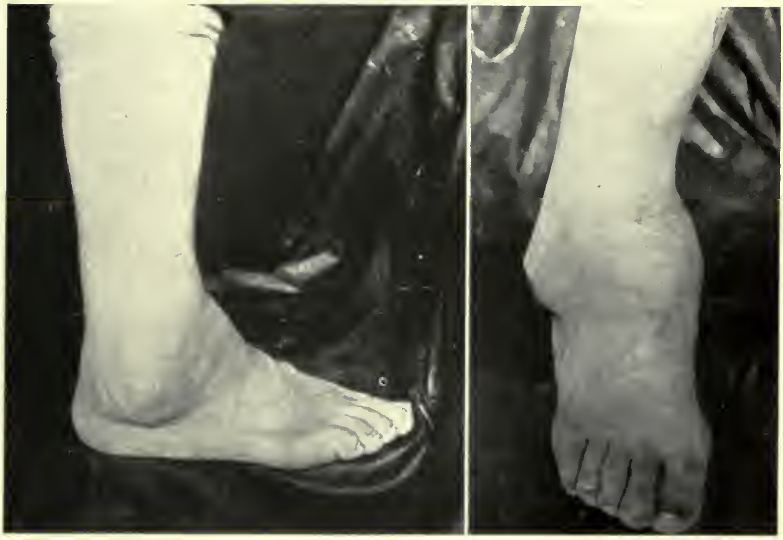
Here is another fierce case. It is one we would like to run away from, but the patient would not consent. When did the patient first notice there was pain in the right ankle?

INTERNE: About ten weeks after the fracture.

DR. MURPHY: What made you think he had a fracture?

INTERNE: Well, it was a trauma.

DR. MURPHY: What symptoms did he have which would lead you to believe that he had a fracture?



Figs. 15 and 16.—Case of Charcot ankle, showing deformity before operation.

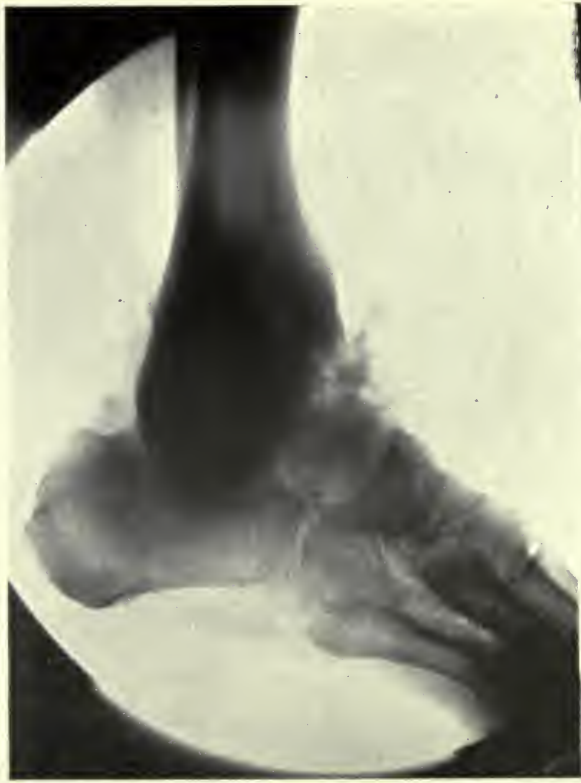


Fig. 17.—Skiagram showing Charcot ankle-joint.

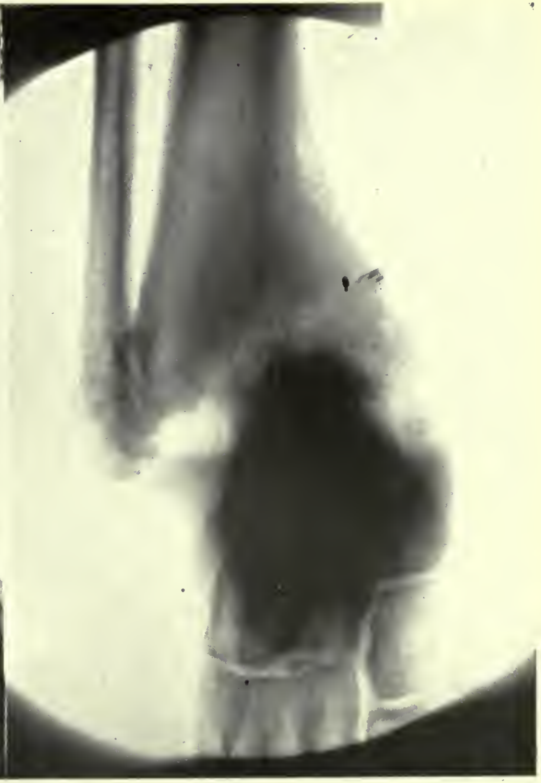


Fig. 18.—Skiagram showing Charcot ankle-joint. Other angle.

INTERNE: Well, the doctor pronounced it a fracture of the right ankle.

DR. MURPHY: That is indefinite. On what basis did the doctor make his diagnosis?

INTERNE: I think he based it on the deformity and inability to use the foot.

DR. MURPHY: What else?

INTERNE: The pain and swelling.

DR. MURPHY: The *pain* is what he did not have. That is the kind of diagnosis doctors make in the library, when they are sitting in an easy chair and are feeling comfortable. That is exactly what he did not have. The reason I wish to accentuate it is that the doctor who had this case has been rather severely criticized, and the patient, when he came to us, had a very vindictive feeling toward the doctor over the evil result, and his doctor had nothing whatever to do with the result. It shows how cautious we have to be in passing an opinion on the work of others. This was a Pott's fracture originally. When the primary dressing was taken off, the patient stated that the bone was in perfect apposition, and that I know was true. But the patient kept getting worse and worse until it is in the condition that it is in now, and you say naturally, when you look at this ankle, it is a bad result from a Pott's fracture. No, you never have *inversion* of the foot in a bad result from Pott's fracture. It is always an *eversion* of the foot when there is a bad result from a Pott's fracture. This man has inversion of the foot. The photograph shows that the foot is displaced inward, not outward. This means that some other lesion occurred in the ankle, and that your doctor's desk-dream diagnosis is wrong. When I asked this man how much the ankle hurt him when he walked, he replied, "Doctor, it never hurt me a particle when I broke it." That tells the story as to what is the matter: that it is a syphilitic joint or tabetic limb; these patients usually have no pain at the time of fracture. It is entirely a Charcot joint following a fracture in a tabetic limb, and the key to the diagnosis was *no pain.* You see where the interne made a mistake. If there were deformity and inability to use the foot, it is all right, but the record should show the symptoms of fracture as they were in this case, and should accentuate the absence of pain.

This is an unpleasant case to operate on, since he has all the symptoms of tabes. He has had injections of salvarsan. He has the Argyll-Robertson pupil, absence of reflexes, except that he has no tabetic gait. The interne was misguided because his patient had no ataxia in his walk. This case is of double interest to us because I came near slipping up in the diagnosis of a case that preceded this. The previous patient, while riding a bicycle, was thrown, and immediately remounted, rode to his place of business, and worked for six weeks thereafter. At this time there was enormous enlargement of the hip-joint up over the ilium—so much that I thought it was a sarcoma. The *x*-ray, which was defective, did not indicate that there was a fracture of the neck. He had worked every day from the time of the injury, which was a period of six weeks. After I looked him over carefully and saw the great swelling I said there is a callus on the side extending up from the side, and that it was a sarcoma undoubtedly, as it came on in six weeks; but when the patient was going out of the office I said to him, "Why do you limp so much? does it pain?" He replied, "No, doctor, it does not pain at all. It has not pained me from the day I hurt it." That told the story. I called him back and changed my diagnosis to Charcot hip. A subsequent *x*-ray made by Dr. Hochrein showed a fracture of the neck and great ossification of the periarticular fibrous tissue extending up into the muscles almost to the crest of the ilium and to the pectineal line in front.

The patient before us has a little motion in his ankle, but not much. Most of his motion is undoubtedly from his astragalus and os calcis, so that preservation of this little motion in the astragalotibial articulation is not important. We expect to obliterate this. But what we must try and do is to get the astragalus far enough out to be in the middle of that tibia, so that we can get a good leg for him to walk on. We must take out this enormous callus that is here and endeavor to preserve the limb. There is the tendon of his tibialis anticus, so that I can make my incision along there and get a good portion of this out. I cannot elongate the muscles or tendons attached to the foot. You can see where the line must be. I must take my tibia and fibula off on this line, and my astragalus off on that line, because if I elongate I will put too much tension on the posterior tibial artery where it comes around the curve and compress it, and we will get gangrene of the foot. Mechanically, there seems to be no limitation to what you can do in this line of work. Tabetic fractures unite, as a rule. I remember very well an experience I had with a patient of mine whom I met in Europe. I left him one evening, and in five minutes was called to his room. He was a tabetic, and in pulling off his shoe fractured his thigh. The limb was put up in a plaster cast, he was taken to his home, and he got union, showing how these fractures unite.

There is an excessive reproduction of bone in the fibrous tissue of the capsule of the joint. It is one of the processes that occurs in connection with an embryologic rule, and it follows an embryologic element. There is in the embryo the production of bone in the definite fibrous layer in the mesoblast; that fibrous layer produces all the bones except the bones of the face. In the face you have an embryologic layer in the deep portion of the branchial cleft, and in that are produced the bones of the face. Here in the capsule of the joint and around the joint you have left the original portion of the embryologic layer which existed in the embryo. Remember, you have no joint originally. You have a continuation of bone, or a continuation of this embryologic membrane that produces bone. When the joints commence to form, they do so first as a cartilaginous mass, then a little streak or line between the two bones is formed. When that streak commences to enlarge, it forms a line across the cartilage, with no membrane between the cartilages; then you have a separation of the cartilage and the formation of the membrane, and you have a straight fissure making up the joint.

There is the beginning of the line of exostosis. It is exactly the same as you see it in that picture. I am going to take out that mass, and I shall adhere as closely to that line in its removal as I can. I try to estimate how much of this mass is produced from the tibia and how much from the astragalus. Most of the bone was produced from the external malleolus; some of it was produced from the periosteum of the astragalus. I am taking off some of the fragments from the periosteum of the astragalus. I am trying to escape the peroneal group of muscles here in my dissection. I am endeavoring to leave the periosteum. This fibula is thrown out from its normal relationship to the tibia. I am going to endeavor to get this point of the external malleolus and put it close to the astragalus, where it belongs. Here is an enormous external callus which has been pushed away out of position. There is the continuation of the fibula. There is the line at which it appears the fibula was broken. There is an interposing mass that has come in. There is the tibia again. Here is the fibula away out here. I cannot just make out where the tibiofibular articulation is, but I take it that it is right along here somewhere, because there is the tip of the malleolus. It seems to me, both of these bones have united. I take that out, fracturing the fibula, so it can come in, and make a splint for the ankle-joint. I want to keep his malleolus. There is the articular surface. Here is the tip of the malleolus. I am going to see if I can take that out there and bring these two together again. I am separating the fibula from this deposit. I am able to get this sequestrum out of the field. There is the fibrous tissue, and I will let it stay to reproduce bone. That leads into the capsule of the joint. This is one of the tissues that becomes involved in this spirochetic disease and takes on a metaplasic state, that is, a reversion to the original tendency in embryo for the production of bone. Here is a piece of bone which I must take out before I refracture the fibula. I have this piece split off, and I am trying to get it out without injuring the soft parts. We will take off the tip of the fibula, which projects too low down after the shortening that has been produced above. There is the lesion of the fibula with its thickening. We will take off this lower half. I fracture the fibula above, turn it in, and make it support the astragalus at that point. The next step is to bring the foot out in proper relations of the tibia with the astragalus. As it appears to us now, I think we are going to get a serviceable foot for this patient, with a bony ankylosis. The articular surface is bared for union. In a better subject I would make an artificial joint, but that is not the right thing to do in this tabetic patient.

Let the record show that there was an enormous exostosis on the lower end of the tibia; that there was internal luxation of the astragalus beyond a plumb-line drawn through the inner margin of the tibia; that approximating and abutting on that was a large mass of callus produced from the periosteum of the astragalus. There was a large deposit of bone from the periosteum anterior to the articular surface of the tibia, proceeding from the periosteum of the tibia. The fibula was exposed, and it was found there was a large mass of bony tissue produced from the anterior surface on the fibular side of the tibiofibular articulation, the bone coming from the periosteum and fibrous tissue at that articulation, but mostly from the periosteum of the tibia. This and the fibular surface of the tibia was all removed, and a large mass of bone taken from the external and posterior margin of the tibia close to and articulating with the fibula. The fibula was not changed in its conformation, but it passed downward over the astragalus through the absorption and displacement upward of the articular end or surface of the tibia. It was freed, and an inch taken from its lower end. After shortening the fibula it was fractured three inches above its end, so as to permit the inversion of the tip of the fibula to contact with the freshened bony surface of the astragalus. A large portion of the lower end of the tibia was then removed, so as to level off its lower end and bring the internal articular surface up to the level of the external articular surface, where the greatest absorption had taken place.

When these were all leveled up, it was found that the astragalus was about 1½ inches inward from the plumb-line through the center of the tibia. The leg was then tracted, and it was luxated outward until the external surface of the astragalus articulated with the freshened internal surface of the external malleolus. A 16-penny nail was then driven from the sole of the foot through the astragalus and through the tibia from below upward, fixing the astragalus in a permanent and definite relation to the articular surface of the tibia, now the freshened bony surface. Two 8-penny nails were then driven through the external malleolus transversely through the astragalus. By this time the limb was all fixed in a certain and definite position, with a slight tendency to internal displacement, which we feel was the safest way in which to keep it, considering that we had, or endeavored to produce, a union between the external malleolus and the astragalus, which we feel would certainly maintain the foot in a sufficiently abducted position to be secure and bring it in a plumb-line with the tibia. A few ligatures were then placed, and a subdermal approximation of the capsule made with catgut, and the dermal approximation made with horsehair. There was practically no tension on the flap. The return circulation in the veins of the leg appeared good, making us feel that the direct circulation by the anterior and posterior tibial arteries was sufficient to maintain the vitality of the foot. The technical procedure was extensive, and the work in the removal of the bone or reposition of the foot involved much handling, and, we feel, hazarded the asepsis, but notwithstanding that we have united the entire wound without a drain. There was no special tension on the line of union when the approximation was finally complete, and there was very little blood lost throughout the operation. When the final apposition was made, it appeared to be in good position and secure for a permanent union.

There has been lots of handling of this foot, and notwithstanding the fact that we have been exceedingly careful, now is the accepted time to secure salvation in this case, from a tetanus standpoint, and I shall order that this patient be given 1500 units of antitetanic serum.

Let the record further show that both the anterior and posterior tibial arteries are easily palpated, and both pulsating freely below the field of operation. The future motion in this joint will be between his scaphoid and his astragalus. You will note we have it up to fully right angles, or a little better; that it is dressed at fully right angles or better; that the angle of the foot is at an acute angle with the leg, so that he can go through and get just enough elasticity anterior to that to give him fairly

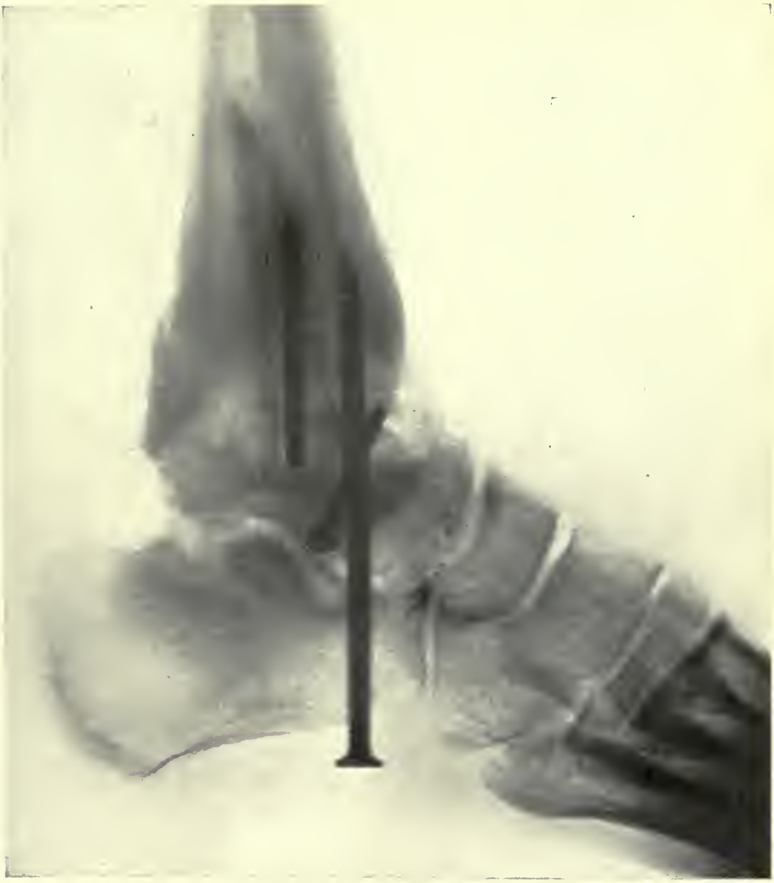


Fig. 19.—Skiagram of case of Charcot ankle taken fifteen days after operation.

good motion. That was where most of his motion was up to the present time.

Let the record further show that there was accurate bony apposition of the astragalus and the internal surface of the fibula, and we expect to have bony union there, *i. e.*, an arthrodesis of his ankle.

Note.—The patient has complete primary union of the soft parts. Five and one-half weeks after the operation the nails in the external malleolus were removed. We endeavored to remove the 16-penny nail, but it was so firmly embedded in the bone, and bony union seemed so secure with it, that it was permitted to remain. All the swelling and edema about the foot have disappeared. There is good bony union. The foot is a little adducted, as we desired it.

UNUNITED FRACTURE OF THE NECK OF THE FEMUR

HISTORY

Merchant, aged forty-one, who was admitted January 2, 1912, because of a non-union of a fracture extending through the neck of the femur. His mother died at thirty-two years of age with spinal meningitis; his father at sixty-eight years, of Bright's disease. Patient had malaria at age of eleven, and has never been ill since. Was married sixteen years ago. Smokes five or six pipes a day. Uses no alcohol. Venereal history negative.

Present Trouble.—February 16, 1910, patient slipped on the ice and fell, striking his hip on the street-car track. He was unable to rise, and was carried home. The physician in charge made a diagnosis of a dislocation of the hip, and the patient was kept in bed for three weeks, during which time he experienced some pain in the right knee and over the sacrum. The patient commenced walking on crutches at the end of four weeks, and discarded his crutches at the end of four months. Since then he has walked with a cane. It does not pain him to walk, but there is a noticeable limp from a 2¼-inch shortening. Also he is unable to flex the thigh upon the abdomen when in a sitting posture, or to cross the injured leg upon the other.

COMMENTS AND OPERATION

The annals of this case are very simple for a complex situation. How the error of luxation occurred we do not know. We do not know what led the doctor to make that diagnosis. I presume that he put traction on the leg, that he felt something snap, and thought he had reduced the luxation. In place of that, he merely had the fragments slide by each other and had the jar that goes with the sliding. There is considerable upward displacement. There is one thing that is annoying me quite a little, and that is in the *x*-ray there is an apparent fragmentation of the head. It looks like a fragmentation or a dissolution of the head of the femur. That is not pleasant. You see the great shortening this man has. You can take his leg and pull it down about 1½ inches. I find that he has 3 inches of shortening without traction, and 1½ inches of shortening with traction. It shows the latitude of horizontal motion at the hip which he has. There is a crease in the head. I do not like that. I am afraid when I get in there I may not find the head there at all. If I do not, I shall have to make a head from the trochanter. I shall have to cut off a good portion of the trochanter, leaving enough for the attachment of the gluteal group of muscles, and put the trochanter on the joint end of the neck. This I think is the feasible thing to do. If we cannot find the head, we will take this portion of the trochanter, swing it round, and put it into the acetabulum and endeavor to get a union. We did that in one case by a plan which we had already figured out, and an emergency arose in which we carried that plan out.

In the patient where we transplanted the fascia lata from the trochanter some eight weeks ago we had no flap we could use at the knee. The patient was operated for tuberculosis, and a long cut had been made in the knee, leaving nothing to utilize as a flap; so we took a flap from the fascia lata and bursa of the trochanter and transplanted it onto the knee. Notwithstanding the fact that the margin of the skin-flap sloughed, we have it all healed in. That is the last step. When you can take tissue from some other position in the body and make a joint with it, where you have already cicatricial tissue, that gives you an opportunity to husband the skin and lateral capsular flaps.

The reproduction of bone, after it has been transplanted, is certain, and that you can transplant the head and make a head from the trochanter is equally certain, because it is based upon the principle of putting the transplanted portion of bone in contact with osteogenetic elements.

The one essential in all these joint cases is that of asepsis,

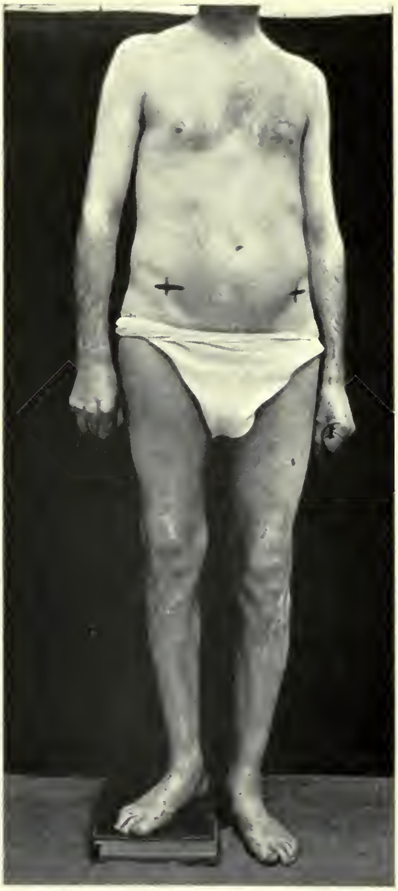


Fig. 20.—Case of ununited fracture of the neck of the femur, showing the marked shortening of the leg.

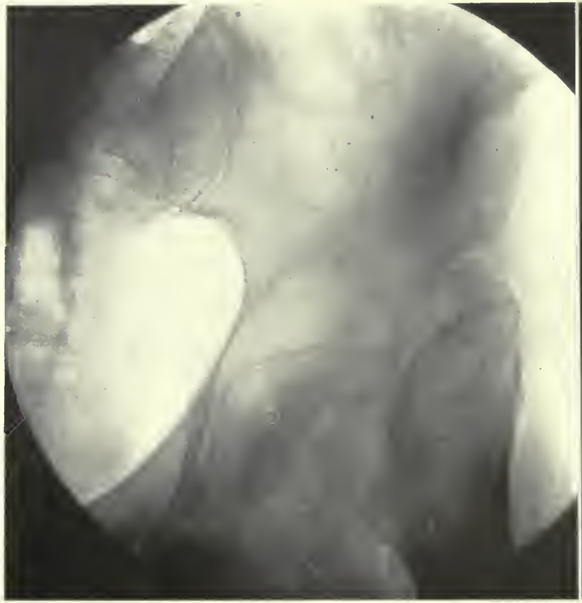


Fig. 21.—Skiagram showing ununited fracture of the femur. Marked upward displacement.

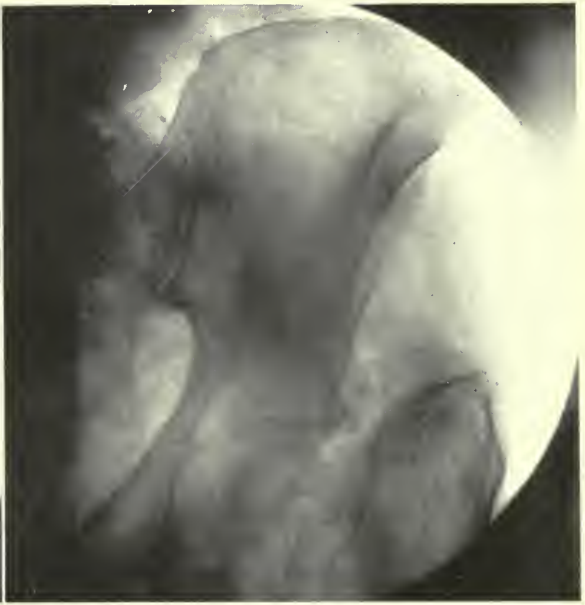


Fig. 22.—Skiagram showing ununited fracture of the neck of the femur. Separation of fragments.

and the joints are the most difficult places in the body to obtain asepsis—particularly the hip-joint.

In the case before us today I am not going to take off the trochanter in the initial steps of the operation, as I usually do, because my treatment of the trochanter will have to depend entirely upon what kind of head I have. If I take off the trochanter, I will have to handle the muscles in a different way, so I will let the trochanter stay and attack the joint from in front. The advantage we have in going into a joint in front is this, that I can leave the trochanter temporarily attached. Ordinarily in this operation I throw the chain-saw around it, saw off the trochanter, and put it out of the field. Here it is different: I may need it. I am going in to the joint in front of the trochanter in place of behind it, and the reason is we have no muscle coming from the pelvic side in front of the joint, which is attached to the great trochanter. The two muscles you have coming from the inner side and passing across that field are the iliacus and psoas, but they go down, you see, and are attached to the lesser trochanter, not to the greater trochanter. If you attack the joint from behind, you come across three muscles, aside from the gluteal group, and must divide them in order to get at the line of fracture of the neck of the femur, namely, the two obturators and the pyriformis. They go right through. They are the rotators. I will get in in front, if possible, preserving the trochanter, but I may have to take off a portion of the anterior surface of the trochanter—that is, to make my new head, I will take off that surface of the trochanter down to the point I show you, and bring it around and nail it to the head from the inside out.

We have had a case in which we did that. There, you see [showing skiagram], is the nail, showing that it was nailed from the inside out. This is not a good skiagram, however, but there, you see, is the major portion of the trochanter taken off and nailed to make a head on the neck. The patient had no head at all. He had a bad fracture of the pelvis, with disappearance entirely of the head. The head does not disappear unless you have a fracture of the neck high up and a rupture of the ligamentum teres in addition to a fracture of the pelvis, permitting the circulation to go into the acetabulum. You see, with a fracture of the acetabulum you get a rich capillary circulation pouring in during the efforts at regeneration. According to this skiagram, the head looks mighty feeble. It looks either as though vascularization was shut off, or there was partial absorption of the head. You can see it, although there is a dim outline of it, and it looks as though it might be comminuted or a portion of it absorbed. You see how the trochanter has slipped away up, but it can with traction be drawn down, for about 1½ inches below where it is now. The skiagram of Willie D. to my left (Fig. 21) shows the result of a fracture of the neck of the femur in a boy of twelve years when the head had been necrotic in the acetabulum for seven weeks preceding its reunion through nailing. The fracture was below the epiphyseal line. The point whether the osteogenetic layer of the epiphyseal line will regenerate and continue to grow will be determined when this boy grows up, and whether the osteogenetic layer in the upper part of that will live. If that lives, it means we can transplant an osteogenetic zone of bone at the epiphyseal line and have it regenerate bone, so that in addition to its acting merely as a scaffolding, there is sufficient regenerative power for the reproduction of bone at the epiphyseal line again to cause the bone to grow in its long axis. That is very desirable, because we get so many young children for operation, both with tuberculosis and other lesions, and we hestitate to operate on them when young, because if we disturb the epiphyseal line, the bone ceases to grow; it will grow in its circumference, but not in its long axis. If that part regenerates, then we can grow bone of the same shape and length of the opposite limb.

One of the most annoying features in this class of work is where there is excessive bone produced around the line of fracture. In many cases we do not have enough bone, but in some cases, when you have union, you have an enormous quantity of bone produced around the line of fracture.

I make only the anterior half of my U-shaped incision. I may not make the other half at all; it will depend on what we have to do with this trochanter. There is the line of muscle we split parallel to its long fibers, and that which I show you now is the anterior margin of the median gluteal. You can see how nicely I am following the line of cleavage right into this joint. I have not made a single muscle division so far. Now we are onto the capsule, following the line of cleavage. Now I am in the joint. There is the neck, and there is the upper portion of the capsule attached to the neck of the trochanter. I think I have a fairly acceptable head. I am going to take off this trochanter now with the muscles attached. There is the fascia we use for transplantation in making the new joint. In turning that around, it is the regular way for doing the operation. We feel we have sufficient head to utilize, and we make a large trochanteric flap. I thought of taking out this portion and of making a new head, but I do not expect to do that the way it appears inside now. We are going to see why union did not take place in this case. There is the head now. See it! It is living! There is the line of demarcation and here is the joint. I will have to obstruct your view for a moment, gentlemen, until I get the head freshened.

What I want to call your attention to is that a portion of the capsule has folded in between the fractured ends of bone, and that is why this patient did not have union. The capsule is united clear across the middle, showing why we do not get union in this case, or in these cases, even in young people. There is the whole capsule of the joint, which I will push out of the field, and I will freshen the fractured surface of the head. Now you see the fresh bone. We have taken the trochanter off at the point I show you, and this is all that is left of the neck. This shows what an amount of absorption takes place in the neck in the direction of the trochanter after a fracture. I show you the trochanter minor, and you see very plainly what is left now of the neck. That is all covered with fibrous tissue, and we must freshen that and swing it around. We are getting the freshened surface down where it belongs. Here you see the lower surface of the trochanter. We must throw all that out of the operative field. Here is the raw surface in front. The whole secret of this is to get the fibrous capsule out of the field. Those who are near enough can see the freshened head of the bone. There is the fascia lata as it fastens up onto the trochanter. I split the muscle parallel with the axis of its fibers and restore it, so that there will be no dead space there.

If all fractures of the neck of the femur were dressed in the way we shall show you, we would get much better results than we do now. It does not matter what dressing you put on, or how you handle it in the beginning, the fracture in such cases as this would not unite because the capsule interposes itself between the two ends of the bone, causing failure of union. All these cases of non-union do not have the interposition of capsule as a cause. I will say this, however, that of our last five cases, four had the interposition of capsule between the ends of the bone. That is an element which is not sufficiently accentuated in our surgical text-books. It has been overlooked, but it is nevertheless true. In this case the capsule was spread right across between the ends of the bone and union was impossible, no matter what dressing was put on.

The surfaces are now freshened; extension and abduction of the limb are made by the assistant; the fractured surfaces are now, you see, in accurate apposition. Two 12-penny nails are driven from the trochanteric side through the neck, and well into the head. They should be at different angles, and about half an inch apart; one nail will not suffice, as the head could rotate on it; with two it cannot. The trochanter is now nailed back in position, the fascia lata reunited, and the skin adjusted with horsehair. No drain. A large carbolic gauze dressing is applied. He is now placed in the travois double abductor splint.

This is an ideal dressing for a fracture of the neck of the femur. It is the simplest kind. In a primary fracture, in addition to this dressing, you have one other necessary element, and that is a 25-pound Buck's extension on the fractured side. We do not need that extension here, as I have the fractured portion nailed in position. I want the edges to press just as hard as they can possibly be pressed together, and that is why I dressed the limb

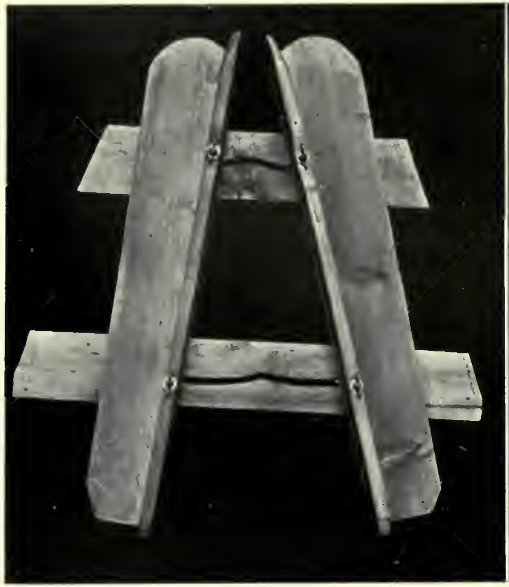


Fig. 23.—Rainey splint referred to in case of ununited fracture of the neck of the femur. Front view.

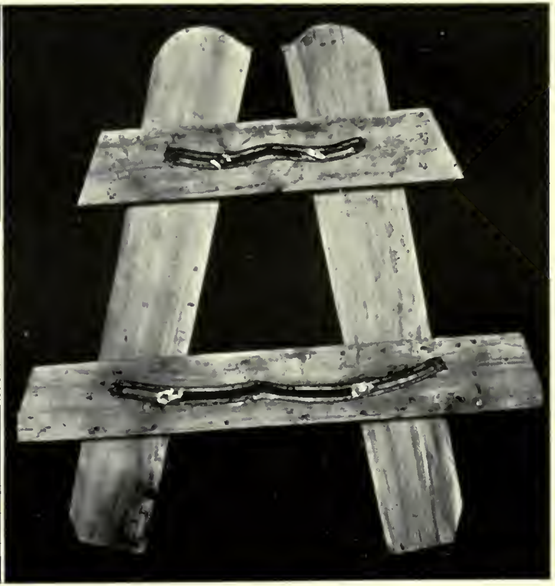


Fig. 24.—Rainey splint. Back view.

in abduction, because in doing that we will get a maximum pressure inward.

The three elements in every fracture of the neck of the femur are, first, the tendency to shortening, which must be overcome by extension; second, rotation; third, the forcing of a continued and permanent apposition of the fractured surfaces, producing abduction of both limbs. One limb abduction is no abduction at all. If this splint were made with one wing instead of two wings, the patient would not get the necessary abduction. When you put in the two wings his pelvis cannot change, and that is the key to the advantage of the Rainey splint. Rainey was my interne and worked out this simple splint for the maintenance of the double abduction principle which I was applying.

Let the records show that a portion of the capsule was found intervening between the shortened and soft neck and head; that the head was broken off just on a level with the margin of the acetabulum; that it had been covered over with cartilage between the segment of capsule that was interposed; that the proximal portion of the neck was also covered with cartilage. A semilunar flap was made, and not the U-shaped—only the anterior half of the U. An incision was made in the fascia lata over the trochanter; the trochanter was separated, and it was demonstrated that there was a head in the acetabulum. The other end of the head was freshened with a chisel, and the inner portion of the neck also. Two nails were used to fix the head to the neck, and the trochanter was nailed back in the usual way.

ARTHRITIS OF THE KNEE-JOINT

HISTORY

MALE; age, thirty-eight; occupation, night caller in train-despatcher's office. Single. Drinks three cups of coffee a day. Smokes about ten pipes of tobacco a day. Uses no alcohol. Family history negative. Has had the ordinary diseases of childhood. Typhoid, twenty-four years ago. No scarlet, pneumonia, diphtheria, or jaundice; no rheumatism or tonsillitis. Amputation of left leg at upper third twenty years ago. Was in bed three weeks. No drainage-tube used. Denies any venereal infection.

Present History.—Dates back to first week in April, 1911. Does not remember exact date. Patient states that as he arose from chair and started to walk, he noticed a slight pain in right knee, but paid no attention to it until a few hours later, when he stopped work to look at it, and noticed that it was slightly swollen. It was not red or inflamed, only seemed larger than the other knee. Pain was not severe enough to stop him from working. Says that he noticed it most when walking upstairs, stepping into a buggy, or taking a misstep at night and striking a little hard, when it would give him severe pain. States that he has had no chills or fever at any time before or during the trouble.

There is no history of any infection. This was the only joint involved. Never has had swelling in any of his joints until present attack. Has not overworked or been exposed in any way previous to onset of present trouble. Swelling and pain increased, and four days after April 1, 1911 (onset), he consulted a physician, who aspirated a clear, watery fluid from joint and injected clear fluid into joint. Patient did not go to bed after injection, but returned home and resumed his duties. Says that during May and June he worked about fifteen to sixteen hours a day. On June 20, 1911, the knee began to pain and swell as before, only this time he thinks it was worse than during the first attack. It became so bad that he was forced to abandon his duties at the end of five days,—June 25, 1911,—and has not worked since that time. Between June 25, 1911, and August 1, 1911, he was not able to walk or touch the right leg to the ground during this period. It also became flexed, so that he was unable to straighten it. Also experienced a grating sensation in joint when he moved it. He was in bed nearly all of July. From August 1, 1911, until the present time he has been able to walk short distances—about a block at a time. The limb still remains slightly flexed, and there is not much change in the swelling. During the whole course of the trouble he says that he has never had chills or fever; that knee has been swollen since onset, but that it has never been red or inflamed; that his appetite has become poor, and that he has lost about 40 pounds during the course of the disease. No day-sweats (night laborer). He has never had any pain when limb was at rest, only when moving or walking on it. Pain has always been limited to right knee-joint.

COMMENTS AND OPERATION

This patient gave a typical history. He had a trauma, and following it he had a locking of his joint, meaning he had luxation of the semilunar cartilage. But he has two additional lesions. He has a typical positive Wassermann, and he has a positive tuberculin, with a history of tuberculosis. Ordinarily, if this patient had neither of these lesions, what would be the indication? The indication would be to remove his semilunar cartilage, because he is a laboring-man and wants to go right to work. But no surgeon with an experience would open a knee-joint with the possibility of tuberculosis or a syphilitic infection being present and active. The opening might not close, and if it did not close, you would have a serious condition of affairs. You could get a mixed infection, then the patient would lose his joint, which would be more serious than his dislocated semilunar cartilage. Following the trouble we have described he began to have an effusion. He has had injections of salvarsan. He will receive a tuberculin injection tomorrow morning. The effusion has disappeared from the knee. The knee is much better.

I show you now a series of *x*-ray pictures showing the progress of the lesion from October 19th to the present time, December 23d. It looks to me more like tuberculosis than syphilis. It looks as if tuberculosis began in this zone [indicating], and following the trauma it is spreading toward the center. Whichever it is, we will not open the joint, because we would add the possibility of another infection to the one that he has there already. In other words, we will build up his system and strengthen his general resistance against both the spirochetes and the tubercle bacilli. We know that a mixed infection aggravates both of these diseases (syphilis and tuberculosis). Any one who has had experience with such cases knows that is true, and we will wait in this case and see what can be done before we make a radical attack on this joint. He will not be put in a cast, but a Buck's extension with 15 pounds will be applied.

Quite frequently in tuberculosis the *x*-ray gives us very little information, and the same thing may be said with regard to syphilis.

I want to show you the picture of a case of Pott's fracture and also one of syphilis of the tibia, to illustrate the contrast between the two pictures. We have two syphilitic tibias, and I want to point out the contrast between this picture and the usual picture presented by a case of syphilis in the head of the bone. Syphilis in the head usually commences in the epiphyseal line, and extends from the epiphyseal line up and down. Tuberculosis in the adult never begins in the shaft side, but usually on the head side of the epiphyseal line. Syphilis begins in the epiphyseal line. Why? Because that is the active growing position, just the same as it occurs in the periosteum and rarely ever in the compact bone of the shaft.

I want to go back to the original manifestation, the speed with which the effusion came on. The record is against tuberculosis producing any of these early primary symptoms. Remember, tuberculosis never incapacitates before three weeks after a trauma. This patient was incapacitated right away. He had a traumatic synovitis, with luxation of the semilunar cartilage, and with this effusion, later tuberculous, came on. This is the only thing to settle. As I have said, tuberculosis never incapacitates an individual inside of three weeks. It is like measles and scarlet fever, so far as time goes, because tuberculosis is an eruptive disease, whether it occurs in a bone or on the skin. The eruptions occur in the bone, on the mucosa, and on the skin, and when you study tuberculosis, go back to lupus, bring it to the surface, mentally, and look at it. Think of what changes would take place if that were brought out on the surface. Lupus is tuberculosis spread out on the surface, and every phase of tuberculosis you see is similar to lupus involving the different tissues influenced by the circulatory or resistant conditions of that tissue. Sometimes you will see lupus that only advances slowly, with a thick, heavy deposit in the neighborhood, and coffer-damming it and preventing it from spreading. In other cases you will see it eating deeply and rapidly all around, with comparatively little or no resistance offered by the tissues, and you have all grades between those two. You will see the sloughing type,—great big patches eaten all around,—and the process is just the same whether it be a bone, intestine, or lung that is involved. If it is in the peritoneum, you have, what? A maximum resistance. If it is in the meninges, another serous surface, you have a minimum resistance. Tuberculosis of the peritoneum gives the greatest percentage of recoveries of any serous surface. Tuberculosis of the meninges gives the greatest percentage of mortality. What is the difference? It is a difference in the local resistance of these two membranes. This variation of resistance to tuberculosis must be reckoned with in the treatment of every tuberculous focus.

PELVIC TUMOR

HISTORY

The patient, a female, is a school-teacher aged fifty, who was admitted to the hospital December 19, 1911, on account of a tumor mass in the pelvic cavity. Family history: Had a half-sister who died of carcinoma of the breast at the age of thirty-one; an aunt who died of carcinoma of the breast at the age of thirty. These relatives were all on the father's side, and had been closely associated with the patient. Has never been married. Venereal history negative. Menstruation started at the age of seventeen, was of the twenty-eight-day type, and regular; no dysmenorrhea; duration, three days, 5 napkins a day. This continued as described until December, 1910, when she twice had intervals of two months between the flow. In March she stopped menstruating, and did not menstruate again until November 15th, eight months later. Her flow at this time was of three days' duration and scantier than usual. Three weeks later the patient started to menstruate again, two days after a severe fall on her hip. This time she flowed very profusely for seven days; the flow contained many clots, and there was more blood than ever before. This was on December 7, 1911, and she has had no flow since. On this last menstruation the patient experienced some dysmenorrhea.

Present Trouble.—At the age of thirty-one a growth appeared in the right breast. This growth was excised at this time, nineteen years ago. Four years later a growth appeared in the left breast. A radical operation was performed on this breast at that time; the left breast was removed, and the axillary glands dissected out. There has never been a return of the tumor in the left breast since the operation, fifteen years ago. *Thirteen years after* the excision of the tumor on the right breast there was a recurrence in the same breast; this growth was operated, and the breast and axillary glands were removed. Two years later the growth again occurred, this time near the sternum; this was removed six months later, and at this time, in March, 1910, a growth in the pelvic cavity was discovered by the attending physician, who pronounced it a fibroid tumor, but she had no unpleasant symptoms from this. On December 5, 1911, the patient fell, hitting on her right hip; since that time her abdomen has been steadily increasing in size. There have been no subjective symptoms, except a dragging sensation in the abdomen and some backache.

Internal examination, December 19, 1911, shows a mass filling the left fornix; the cervix is displaced to the right and the fundus to the same direction, but a little backward. The mass is continuous with the cervix, and of wood-hard consistence. The cervix rests on the right side of the mass, which is in the left fornix, and one cannot differentiate between them. The mass moves in the pelvis; there is no infiltration of the pelvic floor to indicate primary malignancy. The abdomen is filled with fluid.

COMMENTS AND OPERATION

This is a complicated case. The patient was admitted to the hospital yesterday morning. I merely saw her for a second or two. We decided not to operate until we found out the condition of her urine, and I am informed the report will be ready by the time the history is completed and read. [History read by interne.]

DR. MURPHY: You have heard the history of this case as read by the interne. What kind was her terminal flow? Was it fully up to normal menstruation?

INTERNE: I do not know.

DR. MURPHY: Was it more than normal? Was it associated with clots? Why do I ask these questions? Because if she had a closing of menstruation with a fibroma, it would be the full quantity right up to the final menstruation, and then cut off abruptly and not return, provided the fibroma is near enough to the mucosa of the uterus to influence menstruation at all. These things should be a part of the history of the case. They are very important.

Didn't she have an injury before the next to the last menstruation?

INTERNE: I think not, doctor.

DR. MURPHY: You are right about that. She had one menstruation after she had an injury. I want the two connected, because there is another element in the history of this case that we ought to clear up. According to the history, she never had pain with menstruation before. Here comes in a new rôle in this case. In the first place, she had a short menstruation after an interval of eight months, which was associated with trauma, and within three weeks the severe trauma was followed by uterine hemorrhage. A woman who is now in the menopause should not have that kind of hemorrhage from that kind of trauma. That part of the history does not fit. Let us see why it does not fit. [Here the interne reread the history of the present trouble.]

DR. MURPHY: And the abdomen was full of fluid?

INTERNE: Yes, sir.

DR. MURPHY: And this fluid has all come on since the fall or injury?

INTERNE: Yes, sir.

DR. MURPHY: I examined this patient a year and a half ago, but no record was made of that examination. I advised her at that time to have an operation performed for a lesion she had in her left pelvis. What it was I have not the slightest recollection. I make no attempt to keep the essentials of these cases in mind until they come to my office; there I have a record made of every case, and the essential features accurately noted.

She came here in the forty-ninth year of her age, menstruating regularly, without an increase in the flow. She went along and skipped eight months. She had menstruated before the trauma, and following the trauma she had profuse menstruation. She had a flooding, so to speak, with clots. Following that she began to feel a tumor in her abdomen, although the injury was on the opposite side to where the tumor was situated. In my examination I located the tumor on the left side of the pelvis.

The history is further complicated by the fact that she has had three operations for malignant lesion of the breast, and that now she has fluid in her abdomen.

We have had here, in the last three weeks, three cases with fluid in the abdomen occurring with a history of previous lesions, and these are mighty interesting cases, and, acting on the history of a previous lesion, one always commences to think as to what that means when you find fluid in the abdomen following an old lesion. The first patient, a woman, gave a history of tumor in the left side of her pelvis for a long period of time. Suddenly the abdomen began to swell, without manifestations of severe pain, nausea or vomiting, elevation of temperature, or injury—meaning what? The most common cause for this sudden increase in the size of the abdomen in a case of tumor is a twisting of the pedicle or rupture of the sac; but that is always associated with pain, nausea and vomiting, elevation of temperature, and soreness. An acute attack producing a strangulation sufficient to obstruct the venous circulation of a tumor is usually sufficient to give these acute manifestations. We made a diagnosis of malignancy, and it was a malignant papilloma of the ovary which had ruptured and had spread until there were multiple papillomata all over the peritoneal cavity. This was No. 1.

No. 2 was the case of a woman who gave a history of tumor, but who, without sudden and severe pain, without soreness and a sudden change in her condition, began to have an increase in menstruation. This woman we saw three weeks after the change. She had more pain on the right side following the sudden change in her condition. It was not a sudden change in the typical symptoms of pedicle rotation, but she had a large hard tumor deep in the pelvis which had changed its conformation. It was a pedunculated fibroid, partially rotated on its pedicle, which did not interfere with menstruation. A fibroid that is subperitoneal or pedunculated has no effect at all on menstruation; but intramural and submucous fibroids do influence menstruation enormously.

No. 3. In the third case there was a fibroid with the ruptured capsule and a malignant neoplasm apparently starting. This was due to a two-fifths rotation of the pedicle of the tumor. In even three-fifths rotation of a soft pedicle you can get strangulation of the veins with necrosis of the capsule and protrusion of the fibrous mass through the capsule, resembling sarcoma.

The question that arose in connection with this growth, which had become adherent to the mesentery, was, Was it due to the rotation of the pedicle and interference with the circulation, or was it a beginning sarcoma in a fibroid? That is why I asked for the pathology. This was done at the last operating day. I have not had a pathologic report as yet. I wanted to bring that out in connection with the accumulation of fluid in the abdomen. (The report shows necrosis and not sarcoma.)

In today's case we feel a mass that has the sensation of a fibroid. The cervix passes up as far as you can follow it to the right of the tumor, and then emerges into a firm, hard mass on the left side; but if this woman has a fibroid, it is not close enough to the mucosa of the uterus to have influenced menstruation at all, because in the forty-ninth or fiftieth year of her age she had the menopause. Following that she had another distant menstruation, which is not the history of a fibroid. The fact that she had postponed menstruation up to the fiftieth year, in place of having it at forty-eight, forty-nine, forty-seven, or forty-five, is in itself 50 per cent. in favor of a fibroid, because the characteristics of a fibroid menstruation are these: First, these patients never menstruate in the interim, except it is associated with trauma; second, they flow a longer period of time, and with greater quantity, always at regular menstruation; when they stop, they do not commence to menstruate again until the regular time of the next period. The flow may run from one period into another. When they stop, there is no interim flow, differing from the flow of carcinoma of the uterus. Third, they have the menopause postponed to from the fiftieth to the fifty-fifth year. The cases go on menstruating regularly up to fifty; 65 per cent. of them are cases of fibromata of the uterus. Of those menstruating regularly up to the fifty-fifth year, 85 per cent. of them have fibroma. The influence of fibroma on menstruation is controlled not by its size, but by its proximity to the uterine mucosa. A fibroma or submucous polypus not larger than the little finger may produce more flow than a subperitoneal pedunculated fibroid the size of one's hat. The menopause is postponed, so that when these patients reach the age of fifty-five, the chances are 85 out of 100 that they have a fibroma on that evidence alone.

In this case we have the additional history that this woman had, in all human probability, a malignant lesion of her breast, and not a fibroid lesion. From the report of the examination it is probably a malignant lesion and not a fibroma. If this is a malignant condition of a sarcomatous type, the fluid in the abdomen should, according to the rule, be bloody. In the cases where you get bloody fluid in a serous sac it is pathognomonic of malignancy; if it be not associated with trauma, whether in the tunica vaginalis, pleura, or peritoneum, it does not matter. The same law applies everywhere. If this abdominal fluid has blood in it, if it is not associated with trauma, we will pronounce it sarcoma, and I think we will find it is malignant.

I find it is peritoneal fluid, and not bloody. It does not show evidences of malignancy, so far as that is concerned, but here are some lesions. We will let this fluid out, in order to make conditions favorable for palpation. This fluid has all come on in three weeks. She has no albumin in the urine. I find the peritoneum is thickened and reddened beyond the usual or ordinary capillary condition in the peritoneal cavity. Here is something that tells the story. I run my finger along, and the first thing I meet is this nodular condition. There is the neoplasm. Let us see if this is a papilloma or a carcinoma. All over the mesentery I find the same type of nodules. Here are the changes that have taken place in this peritoneum. I want to show you the peritoneum, so that you can see the changes that have taken place in it, with the deposits on it. It is hemorrhagic apparently, and these deposits look very much as though there was a rupture of an infective focus. You have a ruptured papillomatous cyst. This larger tumor which I show you is undoubtedly a subserous fundus fibroma. The neoplasm from which all these nodules have proceeded is a neoplasm of the patient's left ovary, a papilloma of the left ovary, which evidently ruptured under trauma. We may speak of it as a malignant papillomatous cyst which ruptured under trauma; then began the new implantations which you see everywhere on the surface, and these are the cause of the accumulation of fluid which has followed the trauma. This works out exactly in consonance with the clinical history. I am sorry I cannot hold up the peritoneum so that you can all see it.

There is no good reason for taking out this fibroid, because she has these nodules all over the peritoneum, and I would certainly hazard her life by doing so. The fibroid is a neutral matter in this connection. It is a subperitoneal fibroid. I can swing it in every position. It has no value out and it does no harm in. The thing to do is—what? We have a malignant lesion. We have a type of papillomata which we call malignant because in the present case it will lead finally to a fatal termination. You will doubtless remember the splendid article written on this subject by Pozzi some years ago. See these papillomatous masses implanted here everywhere! The removal of these is out of the question. I have a piece of omentum here which I think shows this rather well marked, and I am going to remove it for microscopic examination. The omentum is all infiltrated with papillomata.

Now, we have this condition perfectly cleared up. The clinical history and the pathologic findings dovetail well into each other. We will close the peritoneum.

Let us go back to the question of uterine hemorrhage, so as to work that out to its final termination. We have postponement of the menopause, with fibromata, depending on the relation which the fibromata or fibroma bears to the uterine mucosa. Just the same as it controls the quantity of the flow, both are controlled by that element and not by the mere fact that the woman has a fibroid.

This woman has, in addition to a fibroma, a papillomatous cyst in her left side which has undergone malignant changes. You know how these develop. They first develop as a cyst in the epithelial elements of the ovary. You have vegetations or warty growths on the inner surface of the cyst-wall, and these go along and often attain a great size. They become encapsulated; then they rupture through the fibrous layer and the peritoneum that formerly covered it, known as the tunica albuginea. As that spreads out with the wall of the cyst intact it retains the malignant lesion, and I have never seen metastases occurring from malignancy in the encapsulated cyst.

Now, you have heard from the history that this patient received a trauma, and following that she had a rupture of the cyst, and then the malignant deposits escaped into the peritoneal cavity and deposited epithelial malignant grafts all over the surface.

If you remember, Pozzi's paper was read at the World's Fair meeting in St. Louis, in which he accentuated this fact: notwithstanding the fact that he saw the peritoneum covered with these papillomata, which he interpreted at the time as being malignant, they all disappear in an occasional case just as warts disappear—meaning what? That they were merely papillomatous grafts, and not carcinomatous grafts. In other words, when the cyst ruptured, the papilloma had not taken on its malignant character, but was a warty growth, pure and simple, and the grafts were of the same type, and they all disappeared like a crop of warts disappear from the hands, the cause of which no one has as yet explained. Even when they are microscopically malignant, they may be clinically non-malignant.

The element that controlled her last flow was the neoplasm in the pelvis. You have menstruation influenced at the menopausal period, and following the menopausal period by neoplasms in the pelvis—cysts, ovarian fibroids, etc. They produce the same type of irritation that causes irregularity in the flow, the intermenstrual flow, and are the most common causes, except where it is of the carcinomatous type.

Let us contrast the menstruation history of carcinoma of the uterus with fibroma of the uterus. A woman with carcinoma goes along in this way: She menstruates regularly up to forty-four or forty-six, *i. e.*, if she reaches forty-four before she gets carcinoma, and the rule is before she gets carcinomatous hemorrhage she has an interim of absent menstruation of many months or years. When the carcinoma of the uterus comes under the forty-four-year rule, the woman menstruates and the menses increase in quantity, and finally a *sudden profuse intermenstrual* flow appears, larger than the normal flow, which should never come except from miscarriage, trauma, or cancer; that is, an intermenstrual flow at that stage of life. When you have a woman with carcinoma at the age of forty-five or fifty, she will have three or four years', or possibly five years', interim, everything going on perfectly well, but suddenly she has a flow greatly in excess of normal menstruation. That means at the age of fifty, when it is not associated with trauma, that it has 100 per cent. of chances of being a carcinoma, regardless of what you find when you make a vaginal examination.

Next, carcinoma often begins at the cervicocorporal junction, which has a rich vascular supply, and has not eaten through on the vaginal side of the cervix, but is in the cervical canal, or in the neck of the cervix or the neck of the uterus; it cannot be palpated and cannot be diagnosed without careful curetment.

The most common place for the columnar type of carcinoma is in the corporal portion of the cervical canal. These patients will flow profusely; frequently they will flow for a couple of months and then stop; they will again have another flow, or may never again have one.

I want to call your attention to the fact that here is a woman who has passed the menopause, and she has a flow in excess of the normal amount. You must interpret that as indicating malignancy, and if you would accomplish anything, it must be accomplished at this time. There are women beyond fifty years of age who have "a show," a bloody oozing, and they will say to you, "Doctor, I think I am getting my second girlhood." If it is just "a show," never make a diagnosis of carcinoma until you have made a careful examination, and in the enormous percentage of cases you will find that show is not indicative of cancer, but it is the show of senile vaginitis. The slightest touch or friction will cause the mucosa in a woman of that age to bleed more or less. Again, the bleeding in such a case may be due to a small polypus protruding from the cervix. Sometimes a polypus with hemorrhage is associated with pain. The polypus being in the uterus, as contraction of the uterus begins to take place on account of senility, and there is not space enough in the fundus of the uterus for it, the patient has a colicky pain, and then comes the show as the polypus is expelled from the uterus. You introduce your finger and find a protruding polypus, or the polypus may be strangulated, give an odor, and you have a suspicion that it is a carcinoma. Bear in mind that the flow from this type of polypus is never up to the usual quantity of the menstrual flow, so that you see the clinical history of this case aids one in arriving at a conclusion before the abdomen is opened.

Where we have a solid mass, with the sudden appearance of the flow after a trauma, our suspicions as to malignancy are aroused. Such a hemorrhage had no right to come from a fibroma unless it came from the rotation of the pedicle of the tumor. I have seen necrosis of the capsule of a fibroid with protrusion of the tumor, but I never saw a sarcoma beginning up in that zone and spreading out from that zone, shedding the normal capsule of the fibroid. This case, as it appears to us, is for the present a malignant lesion—not a hopeless one, because it is barely possible the papillomatous cyst was not malignant. We will find all through the omentum clumps and deposits of epithelial cells that have no anatomic right in the omentum at all, because the omentum is a mesoblastic product, and has no epithelial cells, but has endothelial cells. The pathologist will find here nests of epithelial cells. I feel certain as to the report which will come back from the pathologist. *The pathologist should get no information from us. Never give a pathologist a report of what you think a tumor is.* Get his weight of evidence as he interprets what he sees; then you add his findings and your clinical experience together and arrive at a diagnosis, but do not influence his judgment by telling him what you think beforehand. The report of the pathologist is merely an adjunct to the clinical means of making a diagnosis and of arriving at a conclusion, and his is not the final court at all. You have a right to differ from the pathologist and to always question his report. He will tell us that he finds epithelial nests all through; that it is undoubtedly a metastatic carcinoma. You say no, and he will be able to tell you what he saw, but you are to interpret its clinical significance and its final prognostic value.

These implants of epithelial cells you see can be what? Papillomata. You see epithelial cells have been thrown out without their basement-membrane, and the way in which you differentiate an epithelial cell of the normal or papillomatous type from an epithelial cell of the carcinomatous type is that the carcinomatous type of epithelial cell has penetrated the basement-membrane and the cells are multiplying in nests or masses below it, while in the papillomatous type they are above the basement membrane. The pathologist is at a great disadvantage, and the final court of appeal is whether or not this patient gets well. If she gets well, it was a ruptured, non-malignant cyst. If she dies, it was a ruptured papillomatous cyst that had undergone malignant degeneration. These grafts are either innocent or malignant. If innocent, they will get well without your material assistance. Is this all clear? This is a very instructive case to me.

Another thing: there was a great diminution in the quantity of urine when the abdomen began to fill. She reported that her urine was examined three weeks ago and was found normal. That is all right as far as it goes, but it does not do here. The report must be made here. What is it now—normal? We find a diminution in quantity of urine down to only a few ounces a day. Taking all the fluid thrown out into the peritoneal cavity into account,—and the peritoneal cavity was filled with a large quantity in a short time,—that was the reason for the diminution.

Let the record show that the peritoneum throughout the entire cavity was studded with small millet-, pea-, and grain-sized deposits extending clear up into the epigastrium; that the omentum here and there was thoroughly infiltrated with these deposits, and resembled a metastatic neoplasm; that a portion of the omentum was removed for the purpose of microscopic examination; that the papilloma was in the right ovary, down behind a movable fibroid tumor; that the left ovarian zone was encapsulated by adhesions; that there were no adhesions in other positions in the peritoneal cavity; that the fibroid was not disturbed, nor was the papillomatous cyst; that the question as to the final diagnosis of whether it is a malignant papilloma, a carcinoma, or whether it is an innocent papilloma must be determined by the clinical history; that the report of the microscopist must be made a part of the record.

We make a record of the grosser pathology at the time the operation is done. If you wait until tomorrow, you may not have the picture clearly in mind. The fluid in the peritoneal cavity was not bloody. This is quite true of papillomatous fluids, which are not frequently bloody, as is the fluid that is associated with sarcoma. That is true also in carcinoma of the lung, in carcinoma of the testicle, and in carcinoma of the intestines, there being a serous fluid associated with these conditions. When you have sarcoma in these viscera, it is common to have the fluid in the serous cavity bloody. When you have sarcoma of the testicle, it is common to have the fluid bloody, while with tuberculosis or syphilis of the testicle you practically never have bloody fluid. Occasionally in the lung or pleura you have bloody fluid with a tuberculosis, but not always.

Note.—We have just received a report from the pathologist on this fibroma as to whether it is malignant or innocent. The pathologist says it is a fibromyoma, and that the rupture was due to necrosis of the capsule when the strangulation occurred. That puts the patient out of danger of recurrence. Primary union and rapid recovery.

UNUNITED FRACTURE OF THE HUMERUS

Transplantation of Bone

HISTORY

Male; age, twenty-two years; single; roundhouse man by occupation. Has lived in Calumet, Mich., all his life. Never had any venereal infection. Had the ordinary childhood diseases, and pneumonia seven years ago.

History of Accident.—On May 4, 1911, was preparing an engine for night shift and was starting a fire, when a turbine stationary engine 86 feet away blew up, and a piece of the outside casing struck him, as he stood in the cab of the locomotive, about 4 inches below shoulder and also on elbow. The elbow was cut and bruised, and a piece of the olecranon taken off. This cut healed by first intention after suturing. The upper wound was two inches long, and the humerus was fractured. The fracture was set and bridged across with a Lane plate, the cut sewed up, and no drain inserted. Arm was not put in cast, but bandaged tight to side, with lower arm at right angles to upper, and plate of asbestos between humerus and ribs to hold it in line. Two weeks after the stitches were removed, and pus began to exude in upper injury, and was dressed every day until pus ceased to flow, one month ago. Incision now closed. Has not had a chill or fever with his condition, and no bone fragments have exuded from upper wound.

Arm has not been out of sling for any time since injury, except to dress, and elbow is now partially stiff.

COMMENTS AND OPERATION

As I remember this case, the patient came in with the Lane plate in position, and I want to call your attention to the splendid apposition produced by this plate. Look at it! Please notice the excellent apposition. You will notice that there is absolute contacting there of the bone. And what has happened? Non-union. Why did he have non-union with this splendid apposition? He had a compound fracture at the time, as I take it from this history. From an experience running over thirty-five years we have been taught the important lesson that we should not wire or nail or plate a compound fracture. If you do, you insure suppuration. If you close it up with a careful preparation of the soft parts, with a careful iodin preparation of the skin, without washing it, or scrubbing it, or soaping it, or alcoholing it, or solutioning it, but in a dry way clean it out and close it up—in a large percentage of the cases of compound fracture you will have immediate primary union of the soft parts, and the case will go on to repair as a simple fracture almost uniformly in 85 per cent. of the cases. Four cases out of five of compound fracture that are immediately wired, nailed, or splinted will suppurate, and a very large percentage of these will have a failure of union. Now, with this colossal experience, observation of over one-third of a century, it is very evident that we should not adopt that practice. If you will remember, in Lane's early article he did not make it clear that these plates were not to be used in this class of cases, but later he did accentuate it. It should be made clear to every one that they are not to be used in cases of *compound fractures*. If we bear this constantly in mind, we will save a long period of time and avoid many evil results, as shown by Lane.

The most common point of non-union of fractures in the entire osseous system is the junction of the upper one-fourth with the lower three-fourths of the humerus, whether it be a simple or a compound fracture.

The next point of frequency of non-union is 3½ to 4½ inches above the tip of the internal malleolus in the tibia, and even when these bones in this case were in such a beautiful apposition as that doctor succeeded in getting these in, he was incapable of stimulating the reproduction of bone to ossification. We will admit that the suppuration probably played a minor rôle, as non-union occurs where there is no suppuration. It occurs where there is good apposition, and it is in such a posi-

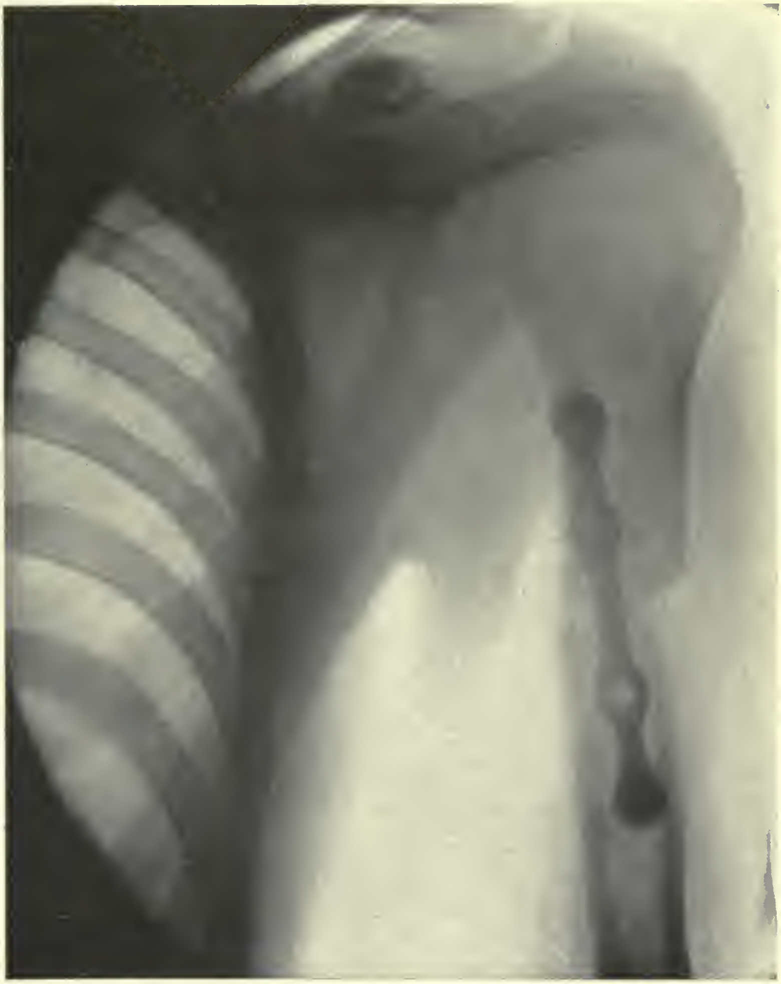


Fig. 25.—Ununited fracture of humerus before operation, showing Lane plate in position without union.

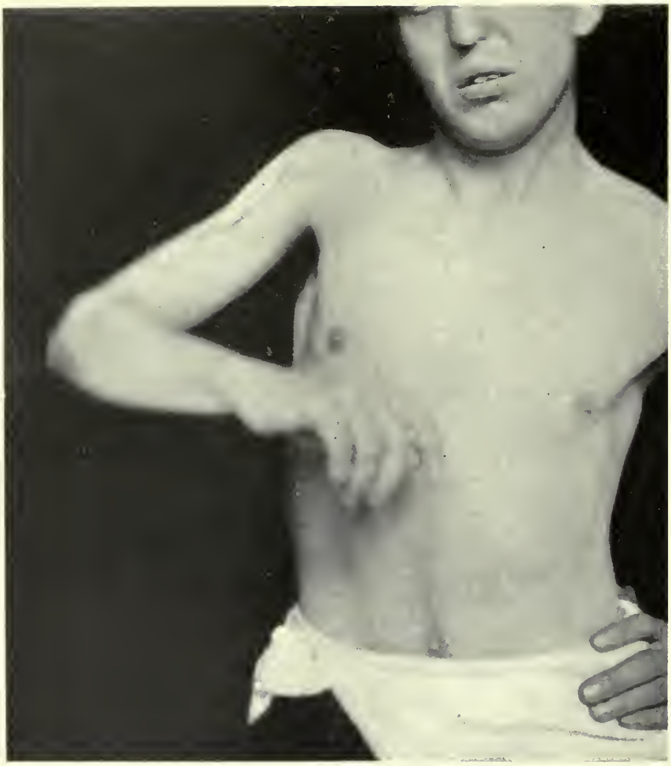


Fig. 26.—Case of ununited fracture of the humerus, showing the uselessness of the arm before operation.

tion that you have a false joint formed, and you can have a joint that occasionally very closely resembles an anatomic joint. Remember, if you open that early you have the hematoma encapsulated. If you open the joint later, you have a fibrous mass with fairly well-developed ligaments, and interposed beween them the cartilaginous end of the bone and a membrane resembling synovial membrane, and a secretion resembling synovial secretion. In other words, you have the production of an artificial, fairly well-formed anatomic joint with ligaments, and with a capsule. It was this condition of things that led me in 1901 to the operation of making artificial joints where we had ankylosis and did not desire it. I reasoned in this way: if nature could make such a good joint and we could look at it and analyze it and examine the conditions under which nature forms a new joint, we are poor mechanics if we could not improve on the crude efforts that result following injury, and on exactly this basis we started the formation of joints, or started the arthroplastic operation that is giving us now such very gratifying and almost uniformly good results without any cases of ankylosis following it.

The element that comes into play here is another one, in that we have non-union probably of the fibrous type, with no tendency on the part of the bone above or below to disseminate or to penetrate through that fibrous tissue with Haversian vessels, and these Haversian vessels carrying forth the osteoblasts which make new bone. You have, in other words, the same condition existing in that bone today that you have where a nerve has been divided and a fibrous neuroma formed between the two ends of the axis-cylinders or axons, and there is no penetration of that fibrous mass by the regenerated axis-cylinders, which is the myelin sheath or neurilemma. The cells of the neurilemma are the ones that must always precede the formation of axons spanning the separation of the nerves. The Haversian vessels are those which precede the carrying in of the osteoblasts to ossify the tissue that is deposited between the ends of the bones. You have the bone regenerated after the penetration with the Haversian capillaries, which capillaries carry the living osteogenetic elements of the bone above and below. Nature is not making the effort, notwithstanding that span is extremely short, and what can we do? We can expose these ends, bring them together, and hope that nature will ossify in the new connective tissue—send its Haversian vessels across; but nature has disappointed us so frequently in this procedure that we do not rely on her longer. We will expose and excavate these ends. We will not depend on nature doing so, and when we have them exposed we will either invaginate one end into the middle of the other, or we will put in a spint of bone taken from some other position in the body. This splint of bone from the patient will form and develop—instead of a contacting material for the Haversian vessels above and below—new bone of the same size and shape as the splint transplanted, and it will give the support that is demanded of the member by the exercise and occupation of the patient.

Here is a case [showing picture] we operated on three weeks ago. You have there a like contacting of bone, exactly as in the other case, but no effort at union. Here we could not very well put in an interosseous splint. All the patient's tendons and muscles on the back of the forearm were divided and separated from ½ to ¾ inch in an automobile accident. We shortened the forearm to bring these together, and invaginated the proximal end ¾ inch into the distal end, securing it, approximating the muscles and tendons, so that we expect to have not only a bony union, but regeneration and reëstablishment of the function of her muscles and tendons. The nerve-trunks are all in front in that position. [Indicating.]

In the case before us we will expose the bone-ends. After exposing them we will decide on which line of procedure we will pursue. There should be a good opportunity for the implantation of a fragment. We sent this man home originally, after a curetment, to get complete healing, so that we are going to have a sterile field into which to insert the implanted portion of bone. If you get suppuration, all this line of work will result in failure. It is more difficult to keep it from suppurating than is the average case that is operated.

You see the line of non-union. This is a place where one must be exceedingly cautious in bone work oft his class. The tissues have a minor resistance. The opportunities for infection are so much greater because you go in and out of the field so frequently.

You will notice that I am taking out the cicatricial tissue because I wish to bring about the most favorable condition for apposition. There is the position of the fibrous union. I am cutting out that fibrous tissue first, so as to get at the line of apposition of these fragments. We are below the attachment of the pectoralis major and above the attachment of the deltoid, which accounts for the position in which this fragment is drawn. Now, we have both of the ends of the bone fairly well exposed. Here is the fibrous mass in between. Here is the bone. I want to show you what it means to be in a position where you have great osteogenetic force as compared with being in a position where you have practically none, and that is in this zone of the upper arm. You will see how nature has splinted that over, and the splendid work which it has accomplished in making a fibrous union.

Let the records show that there is a porosis where one of the Lane screws was inserted. You will see that my instrument falls into it. It has not even filled since the plate was removed. We find an overlapping zone here; we find we have a projection of bone that overlaps ¾ inch, and still there is no effort at union. I think I can lift this up and throw it out. There is a projection of bone, you see. Nature has endeavored to splint it over and failed. There are both of the ends of the bone. One is not yet flush. I throw this end out, so that I can get an opportunity to ream the end and prepare it for the implant. I can make this hole any size that I desire. I am making this hole larger than the reamer makes it to remove the softened zone of the bone plate. The other end to reamed is cut in a similar manner. Now we will take a piece of bone 3½ x ½ x ⅜ x ½ from the crest of the tibia, and I take it with its periosteum. The chisel which we use here is the narrow chisel used by the cabinet-makers. It is a carpenter's or cabinet-maker's chisel. One has to be very careful on account of the age of the patient, because the bone fractures out toward the edge in an elderly patient, but in a young patient you are not so likely to have it split to the surface or to fracture badly at that age. We have tried all kinds of devices for this work, but we have found this chisel very useful, following the plan of getting a little groove to start on and making it deeper with each succeeding passage of the chisel. We do it with less trauma, with less energy, and with greater accuracy. I am leaving this periosteum on, but it will be both a detriment and an advantage. The fact of the matter is that this bone always terminates in the same way. It will always die, no matter how or where you put it in. It plays only a passive rôle for the Haversian vessels. I push it into the upper reamed cavity far enough to permit the lower end to be inserted; then I grasp the fragment with a rectangular bone-cutting forceps, which indents the fragment sufficiently to permit me to drive it far into the lower segment. It is now in the medullary excavations, about equidistant above and below the line of fracture. There is splendid strength we have there now. I can push it up a little farther.

On what now does our result depend? Our asepsis. Everything else amounts to nothing. This is phospho-bronze wire we are using, and it makes a splendid ligature. This is 8-ply wire, which is strong enough. The more plies you have to the size, the safer it is as foreign material. This wire will heal in without a particle of trouble. Wire, like silkworm-gut, stands very little angulation without breaking.

Now, as to union: we have, in the first place, the fibrous tissue at the end of the bone removed; in the second place we have the osteogenetic carrying canals, namely, the Haversian canals, but these Haversian canals in the transplanted portion have no osteogenetic force in themselves at all. All that piece of bone dies, but the Haversian vessels from the medulla and from the compact bony tissue and from the periosteum all start traveling on up through the dead Haversian canals of that transplanted portion.

In the management of these cases from the beginning I will start out in this way and say that every compound fracture should, first, have its neighborhood sterilized, and the method of sterilization which is the best one is the iodin method. There

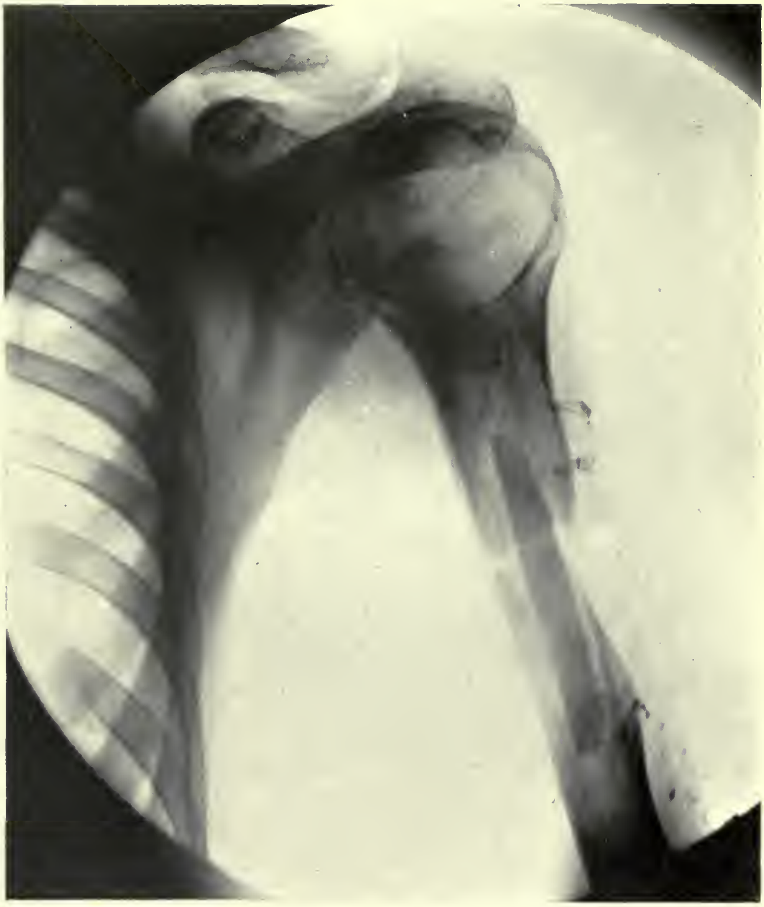


Fig. 26a.—Ununited fracture of the humerus after operation, showing bone splint transplanted from tibia.

is no question but that it is, up to date, the best plan we have. You will notice I am freeing the skin so that it will not dip inward at the point where I wish to approximate it with the horsehair suture, but that it will roll over and its edges come into accurate position. I am keeping outside the line of my catgut suture. You see how much loose skin I have, and what freedom I will have of that skin-flap for approximation. In the second stitch we go near the surface, so as to keep the edge from rotating inward. By putting in one deep stitch and one superficial I make accurate apposition of the epithelial layer, and at the same time get a broad contacting surface. This is one of the places where one must be cautious, or with deep sutures we will have an inversion of and infection from the skin. Just before I started the double suture there was a tendency to inversion. Horsehair is the best of all skin suture material because it is elastic and does not cut like silver wire or silk.

Let the records show that the ends of the bone were exposed; that there was a fibrous connection between the ends of the bone; that they were in the most perfect apposition; that there was an overlapping spiculum on the inner side; that there was no effort whatever at osseous union.

Note.—There was a primary healing of the soft parts with a primary union of both ends, with firm ossification at the end of six weeks.

LENGTHENING OF THE TENDO ACHILLIS

COMMENTS AND OPERATION

This woman has carried her foot in the position you see, so that it looks like a congenital deformity, but it was acquired following a trauma. When you look at the skiagram you will see that the articular end of the tibia rests upon the entire posterior surface of the astragalus, and not on its upper articular surface at all. I have here another skiagram of the case of a young man, twenty years of age, showing the congenital type of this deformity. The tibia rests upon the posterior surface of the astragalus, and not upon the articular surface. In this case we not only elongated the tendon, but divided the capsule of the joint, doing a posterior capsulotomy, and the foot came around with very little force.

Here I expect we will have only to elongate the tendon, and that I will not need to do a capsulotomy. I make the incision on the outer surface, and not over the tendon. There, you see, the tendon is fully exposed. We put a retractor on the other side, and you see how it is divided. It gives you a better exposed field. We split the tendon exactly as we do a nerve, except that we slide it up. There is nothing in front we are in danger of cutting. The tibial artery is away on this side, and there you see the tendo Achillis. We wish to keep the foot from turning in, therefore leave the external half of the os calcis attachment of the tendo Achillis intact, freeing the internal half from the os calcis. We will elongate this tendon 3½ inches. We will divide the external half 2 inches above. The capsule is clearly shown. You see the ends of the tendon. We unite them. These tendons when separated regenerate over a greater span than any other tissue in the body. Three stitches, properly applied, will suffice to hold the tendon ends. I now bend the foot upward. Did you hear the capsule fracture? It was plainly heard by us. The foot, by its own weight, stays in the position in which we have put it, at right angles to the leg. If it were a congenital deformity, I would have to cut the capsule and not break it as I have done. It gives readily because the deformity in this case was simply acquired. After the elongation of the tendo Achillis about 3½ inches, we fractured the fibrous capsule with a minimum amount of pressure, and the foot was flexed and dressed at an acute angle with the leg. The tendons were united with chromicized gut, and the skin closed with horsehair.

Note.—This gave a perfect final result: full voluntary flexion and extension, with no tendency to adduction.

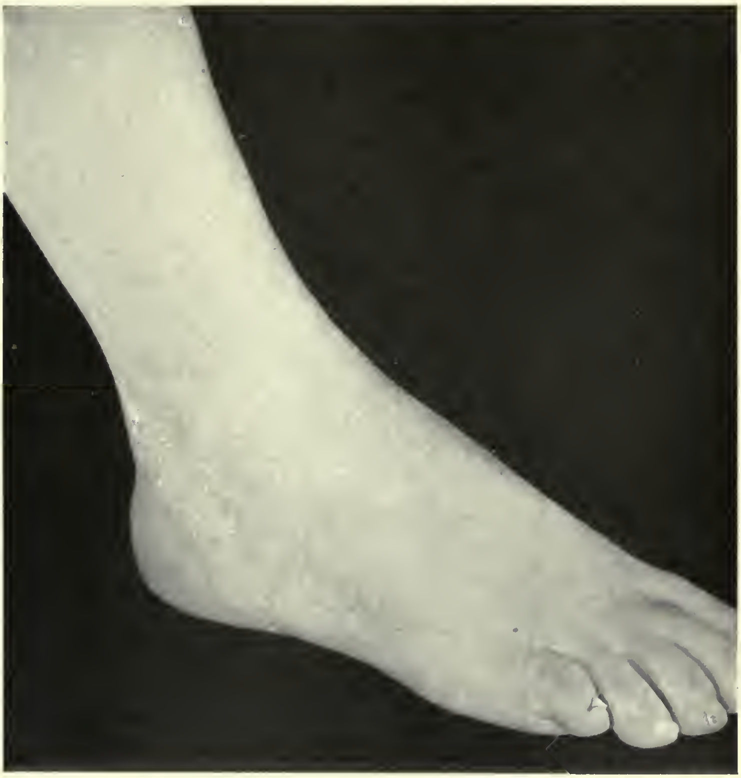


Fig. 27.—Position of foot of patient.

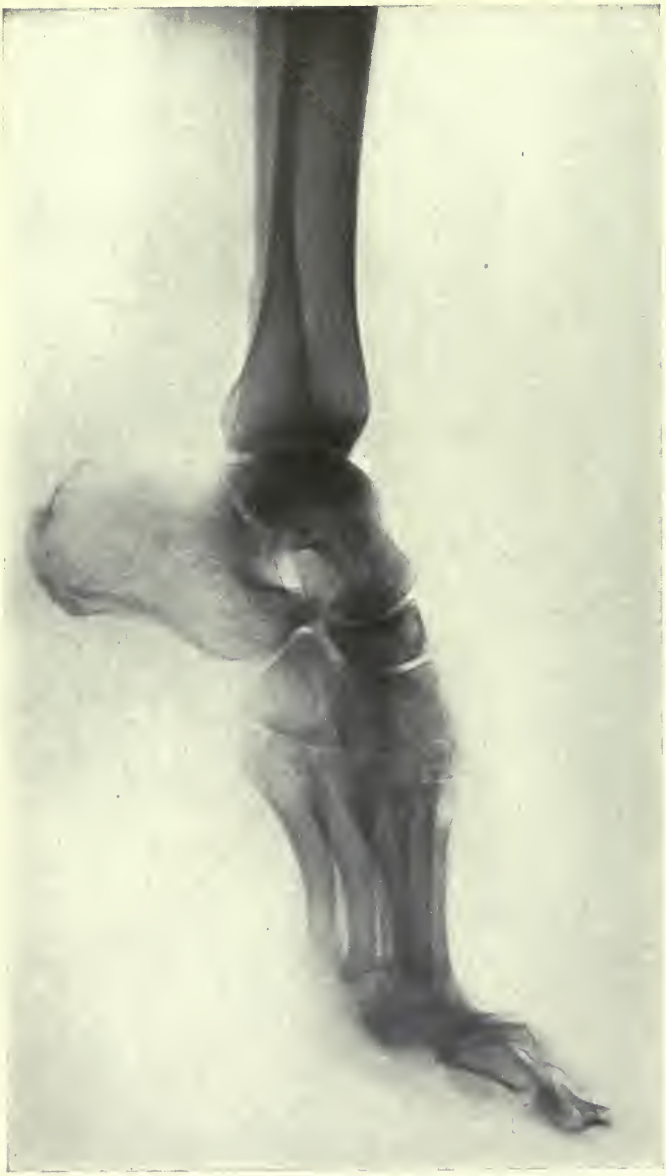
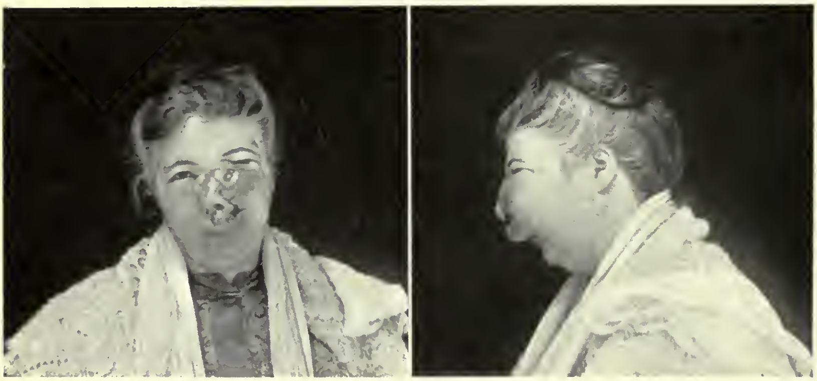


Fig. 28.—Skiagram showing position of bones.



Figs. 29 and 30.—Case of inoperable sarcoma of face. Salvarsan.

INOPERABLE SARCOMA OF THE FACE; SALVARSAN

Here is a patient with a large and deforming sarcoma of the face. We have been using salvarsan with a few good results in just this class of rapidly growing sarcomata, and whether it is the arsenic or a combination of arsenic in salvarsan that does the work, we do not know. In the last fifty years types of tumors, usually sarcomatous in origin, have been cured with arsenic here and there. No one has learned yet which type of sarcoma it is. We have one case now of a woman in Indiana who came in here with an enormous sarcomatous growth infiltrating her pelvic tissues. We could not do anything with it. We did not even make an exploratory operation, because she was operated on once. We put her on daily doses of two grains of sodium cacodylate hypodermatically. It was about two years since that was done. She came in several months after, and at that time I could find nothing in the way of a tumor. She is doing farm work. The two grains of sodium cacodylate is dissolved in 40 drops of distilled water. I do not use the stock preparation, because sodium cacodylate disintegrates very rapidly.

We will give this patient 6 decigrams of salvarsan in 40 c.c. of the oil of sesame, which is absorbed more rapidly than the albolene, because it is a vegetable oil. After the needle is introduced you put on a minus pressure, then you withdraw it. That closes up the tract, and you have no leakage of the material. We give this patient 6 decigrams, 3 on each side of the loin. I repeat this in two weeks or two and one-half weeks, if it shows no effect on the growth. We do not know how fast this will be absorbed.

As to the dosage of the arsenical preparation—sodium cacodylate—that can be given, Dr. Frank Billings has used as much as 6-grain doses in fibrinous pericarditis in children.

Note.—This growth was not influenced in the least by the salvarsan nor by large doses of sodium cacodylate.

CUTANEOUS SYPHILIS; SALVARSAN

The next case gives a positive Wassermann and a positive tuberculin reaction. It is not a tuberculous lesion, but a specific one. This woman has received large doses of sodium cacodylate, and she has had tuberculin. Neither of these agents has cured her. She is here to receive an injection of salvarsan, which we are going to give her today. She came in from Indiana yesterday. She has enormous destruction of her face and head. She has the typical, old-fashioned tertiary lesions of syphilis, the type which formerly did not yield to mercury or to anything, but gradually went on to destruction. I remember very well a case I saw in the Cook County Hospital, when I was an interne, which went on to a fatal termination. The patient had received large doses of the iodids and large doses of mercury, with inunctions, all of which were of no value whatever.

I will give this woman only 5 decigrams of salvarsan on account of her light weight. If you give it in the way originally recommended, you cause much intercostal pain. There is less pain when it is given in the loin than any place we have injected it. We do not use albolene now, nor neutralized alkaline solutions. In three or four cases we have had necrosis of the cellular tissues following the injections of the neutralized alkaline in albolene solutions. These had to be opened and excavated. We have had no other untoward results with salvarsan.

We have had very little experience in the use of salvarsan with the primary lesions of syphilis, because we do not take this class of cases until they come into the grade of being surgical cases.

Let the records show that she was given 5 decigrams of salvarsan with the oil of sesame, and that she will be given $\frac{1}{6}$- to $\frac{1}{4}$-grain doses of the succinimid of mercury hypodermatically daily until the lesions disappear.

Note.—These great ulcers on the face, head, and back healed rapidly and completely under this treatment.

GASTRIC ULCER—SECONDARY OPERATION

HISTORY

Patient is a druggist, aged fifty-three, who returns after a gastro-enterostomy performed nine years ago. The Murphy button was used, and passed out five weeks later.

Present Trouble.—Commenced two years ago in March, 1909, after being completely well for a period of seven years following his operation in 1902. The patient states that about three hours after eating he experienced a dull aching pain over the right upper epigastric region, at the site of the pylorus. This pain was relieved on taking food. However, for the past three months the pain has been constantly present, although somewhat accentuated three or four hours after eating, and slightly relieved on the ingestion of food. He has in the past three months had much epigastric fullness and belching of gas, coming on immediately after eating. His back has pained him a great deal through the sacral region. This is more pronounced on lying down than when sitting up. He has lost 15 pounds in the last four months. His appetite has been poor, and he has lost considerable strength in the past four months.

COMMENTS AND OPERATION

This man has had four laparotomies performed for lesions in his upper abdominal region. What the first three operations were I do not know. I operated on him nine years ago for a gastric ulcer near the pylorus interfering with the outlet of the stomach, and that was the beginning of our differentiation as to when we should and when we should not do a gastro-enterostomy for gastric ulcer, and at that time we discussed the class of cases which were relieved or materially benefited by gastro-enterostomy. This man went along for seven years in perfect health, then he commenced again to have a train of symptoms similar to the regular train of symptoms one finds with gastric ulcer in the neighborhood of the pylorus; that is, pain, eructation of gas, sensitiveness to pressure, distress after food, etc.

Usually, pain is the most common symptom; next, the eructation of large quantities of gas, but this is only secondary to the pain in significance in making a diagnosis of these troubles in the right hypochondrium. With these symptoms we make a diagnosis of ulcer or disturbance with the outlet of the stomach, whether the ulcer or ulcers are on the gastric side of the pylorus or on the duodenal side. Following these symptoms came the slight reduction in weight. In many instances these patients have symptoms, and yet continue to retain their weight without any disturbance. They often retain their weight or gain in weight when a gastric or duodenal ulcer is present. The next symptom of importance is hemorrhage of the acute type, which brings on fainting, nausea, vomiting of or blood in the stools. The hemorrhage may be vomited at any time in the course of gastric ulcer or duodenal ulcer. Blood is found in the stools very commonly, and in all the acute types of cases.

Forty-eight hours ago I saw a patient who for years had been treated for gastric disturbances, who had an ulcer which had apparently healed. He had regained his weight, and even weighed a little more than normally. I saw him five days previous to the incident which I am going to relate now, and he said then, "Doctor, I have a little of my old trouble in the stomach." He had just a little distress,—a little uncomfortable feeling,—but he gave it no more attention at that time. On Thursday—day before yesterday—he went to his work feeling uncomfortable, and fainted on the train going home. He went from the train to be transferred to his house at Highland Park, and on the way vomited an enormous quantity of blood. He also vomited a considerable quantity of blood after getting home, and collapsed, so that he had three hemorrhages to the degree of fainting in one afternoon, with no serious symptoms up to forty-eight hours, and with very little disturbance up to four hours before the manifestation, indicating that he was having an acute attack of gastric ulcer, showing that pain is a relative symptom in these cases, and not the important symptom, as is so commonly met with; moreover, that the disease can go on to the production of a lesion of sufficient severity to give the severe hemorrhages I have mentioned, without the pain being an element of significance at all.

The occult blood in the feces must be examined for with great caution, and must be interpreted with equal caution, because occult blood can be very misleading.

The recurrence of the symptoms after operation has been due to a number of causes. First and most commonly, after the healing of the ulcer the continuance or the reëstablishment of emesis following complete repair does occur, the true interpretation and etiology of which has not yet been brought out to our satisfaction. Some believe it is because the material returns from the jejunum, some that it is due to the short loop, and some think it is due to the long loop, and with all kinds of loops you will have a return of the condition with the opening patent. Second, that the return is due to the closure of the opening. In only one case in which we went in to find the cause of the return of the symptoms have we found occlusion of the opening.

We operated three weeks ago on a patient in whom there was a return of the symptoms, and that accentuates the point that the most common etiology of return of the symptoms is a new ulcer. In this case we showed distinctly—it was the third time the abdomen was opened—ulcers in different places from the former ulcer, the first and second ulcers having healed and the patient getting better in the interim.

In this case I am going through the original scar-line. We will often find a hernial protrusion at the line of union of the abdominal wall, and one can open into that hernial protrusion and injure the bowel unless he is exceedingly careful. I am opening this as cautiously as I can to avoid, as far as possible, an accident. We are below the original incision. I will open the peritoneal cavity and enlarge the opening upward, so as to avoid the likelihood of injuring the intestine. I am making this incision in the usual way—on the slant. We have succeeded in getting in and keeping away from the line of adhesion, so that we are right on the margin of the fixation. There, you see, is the adherent bowel right at the point I show you. I am going to see where these adhesions are, to get the abdomen opened on the side of the greatest fixation. There are adhesions all the way up here from where I am in the abdomen. I feel adhesion on either side. I am separating them with a curved scissors. Now, we are into another open field. There is no free peritoneal cavity. This is the large intestine which is adherent. All this gastropyloric zone is just one solid mass of adhesions. I am going to see if I can get into the peritoneal cavity above to see if there is a free space there. Here is the liver. Here is a free space above. There are no adhesions of the stomach to the liver in the upper zone. There are adhesions in the lower zone. It is fixed in the lower zone. We will throw this large intestinal adhesion out of the field from the right side, so as to free the hepatic flexure of the colon. We shall go in on the central side and determine the condition of the point of anastomosis. These adhesions we have here will all reform, no matter what we do to free them. I used to think that we could cure intestinal adhesions and avoid their reformation, and we have used the omentum in all sorts of plastic ways in the management of them, but, notwithstanding that we have had recurrences of most of the adhesion that we have endeavored to free and to retain free after operative procedures. Whether these are etiologic of the symptoms at the present time we do not know. We are not willing to charge them up on that side of the ledger. Now you can see we have not had the stomach in view at all in any part of this operation.

Let us see how the posterior gastro-enterostomy is. It was made with the button. Here, you see, is the line of union. The gastro-enterostomy was done nine years ago. You see there are no adhesions at all around the line of union. It looks like a normal opening—like a physiologic opening. You will see it was a short loop. There is no loop at all above. Let us determine whether or not this opening is patent. There is the anterior portion of the stomach-wall. My finger is now nicely in the opening, and it shows up. It moves around freely in the opening. There is ample space for my finger to go in to the first joint, without any tension at all. I lift it right up into the field without any tension. There is my finger in the opening on the stomach side running clear through, showing that there is ample opening and there is no closure. That clears up one feature of this trouble, namely, that it is not due to a closure of the opening. That is exactly in consonance with all our cases, with one exception, which is peculiar, namely, that the symptoms have returned. Let us see if we can find out any other reason for these adhesions reforming. We are in the space above the stomach, so that we can get these adhesions free here. These are adhesions, not organic union, in the sense that they are cicatricial following irritation. Let us get to the pyloric zone and see if we have evidences there of another ulcer. Here is the stomach, situated high up, and firm with these adhesions in the line of union in the middle. I want to show you the direction of the adhesions. The pyloric zone is pulled around to the midline, and adherent to the left of the midline, in place of coming down in this direction. There does not seem to be very much, if any, dilatation here. We must free the pylorus if we can. We will see if we can get a proper estimate of the pyloric zone of the stomach. There you see the binding pillar that pulls the pylorus clear across to the midline. That is an adhesion. We divide all these, free the pylorus, and get in at this point.

Now we are palpating the gall-bladder. Let the record show that the gall-bladder is distended but compressible; that it is entirely covered with a mass of adhesions, fixing it to the large intestine and duodenum; that there are no stones in it. Now I am palpating down along the cystic duct. The head of the pancreas is not infiltrated; the common duct is easily located; its wall appears to be a little thicker than normal, accounting for the ease of dilatation, but nothing to indicate infection of the pancreas. There is no stone in it. I am now on the under surface of the pancreas; there is no stone palpable in the pancreatic duct. This man has had no colic at any time, and there is nothing in these tissues that I can see that justifies his present symptoms except a torsion of the pyloric zone of his stomach. I am going to correct that torsion by fastening the stump of that down to the tissue in the neighborhood of the gall-bladder and straighten it. I think I can prevent adhesions from forming in other portions of the stomach in doing that. I am going down close to the gall-bladder and taking that zone and coming on top here, turning the abraded surface of the stomach down into the gall-bladder zone, and covering up this abraded surface of the zone of the stomach so as to rotate the stomach back to its normal relationship. I now tack the great omentum over the anterior wall of the stomach, to protect it from the anterior abdominal wall abrasion. He has no evidences at all of an existing ulcer now. His ulcer is entirely healed, and both his pyloric and his intestinal openings are patent. The symptoms which he had, if you remember at this time, were an ache and distress, but most marked two hours after his meals, when his food wanted to escape from his stomach, which is the time for it to do so in greatest quantity.

I am going to make a catgut stitch on the wall of the stomach, to cover in as much as possible of the abraded zone, and let that rest on his stomach, which is a movable organ. We use just one tacking stitch. This is all raw peritoneal surface here which I show you. Every abrasion in the peritoneal cavity should be covered. I bring all the raw surface into submission, and turn the edge over to the stomach, so as to keep it as near as I can free from adhesions. Now, as much as can be done in the way of covering these abrasions with sutures has been accomplished. I am going to place this raw surface up against the abdominal wall as well as I can, and cover it as well as I can with an additional tack of omentum. We will make as much of an ectropion of that cut edge of the peritoneum as we can under these circumstances, using all the surface that is here to get the line of approximation, and lessening as much as possible future fixation. From this on we can get a fairly good ectropion of the cut surface of the peritoneum.

Let the records show that we believe the etiology of the distress was a rotation of the pyloric zone of the stomach, turning sharply upward and to the left, and fixing it to the anterior abdominal wall to the left of the midline, and fixing it in an immovable way; that the adhesions were firm, inelastic, and had to be divided. Let the records further show that at the completion of the separation an examination was made of the gall-bladder, and it was found entirely embedded in adhesions; it was compressible; it contained no gall-stones. There were no evidences of obstruction of either the common, cystic, or hepatic duct in any part of the clinical course, nor were there any evidences of a foreign body present in these at the examination. The pancreas was not in the least infiltrated, nor was there evidence of stone in the pancreatic duct from the palpation of the under surface. This palpation was easily made.

Note.—Much immediate relief followed the operation, but this is no criterion as to the permanency of the relief.

ANKYLOSIS OF THE KNEE—ARTHROPLASTY

HISTORY

MALE; age, thirty-three; single; farmer by occupation. Admitted September 21, 1911. Has lived in Iowa all his life. Family history negative. Had the ordinary childhood diseases, with good recovery. Never had typhoid, pneumonia, or other adult diseases. No peritonsillar abscesses. No alveolar abscesses or ulcerations. Denies venereal infection.

Present History.—Patient says that in the fall of 1901 he began to lose ambition and did not feel as lively as before. Noticed after meals that he would rather lie down than go back to work, and did things half-heartedly. Thinks he may have lost some weight during that time. March, 1902, had swelling, redness, and pain in next to great toe of right foot; four days after had redness, swelling, tenderness, and pain in next to great and middle toe of right foot, and next day in next to little toe of right foot. Heel and tendo Achillis at the same time became tender when the patient would walk around, and patient walked on toes to relieve pains; had no chills or fever, and did not go to bed. A few days after this had pain and tenderness in right leg, just about patella, when pressing over that region or on walking, and with motion, and about same time had pain and tenderness on motion in index-finger of right hand and just above elbow of right arm. Had pain and tenderness on motion along outer side of right thigh about two weeks after onset of trouble, and a catching sensation in hips when walking around after one week. Had pain above knee in left thigh and down calf of left leg, which seemed to be most tender. Up to this time had been up and about, although in misery, but on April 8th went to bed on account of pain on motion. Had chilly sensation and drenching sweats for about two weeks after going to bed; had no appetite and bowels were very loose; could eat nothing fat, not even butter. In the week previous to going to bed, and up to July, had a temperature ranging from 101° to 103° F., and one week after going to bed had distinct chill lasting five minutes and temperature of 101° F. Legs at this time painful, and both knees and hips painful, but not very much. Had massage at this time, which only increased the tenderness and pain and caused the patient to breathe faster and caused distinct consciousness of heart action, which was increased. A month and a half after going to bed legs began to flex on thigh; legs were pulled every day, and patient tried to stand, but could not, on account of pain. Patient says that when he held himself in tension had much less pain. On awakening after sleep, when muscles were all relaxed, the pain would be very severe, and he would contract muscles to ease pain. On the first of July he sat up in a wheel-chair, because pains seemed to have eased off; legs were still contracted, but no stiffness of joints.

July 22d was taken to Colfax, Iowa, on a stretcher, to take baths; legs very tender and painful, and patient was nauseated and very sick the first half-mile on road; after first half-mile severe pain left him. Was there five weeks; his appetite returned and pains eased gradually, but legs were still contracted. Home one week, then to Hot Springs, where he was for nine weeks, during which time the legs straightened out; no pain. Became gradually stronger, and could walk with cane. That winter he gained weight, appetite increased, and he felt nearly normal. In the spring of 1903 had severe pains again in left leg, extending from knee to hip; walked with two canes for two weeks. Pains eased up, and he felt better for one month. Then he began to have pains in both legs, pains increasing in posterior part of thighs, some pain in both knees, and soreness of heels, but walked all this time. In October the legs began to contract again; then he tried osteopathy for three weeks, but was so tender in back and arms and legs that he quit walking. In the spring of 1904 sat up in chair and gradually gained in weight. Contracture remained, but legs were not stiff. In the winter of 1904–05 was fairly well, with very little pain and ate well and

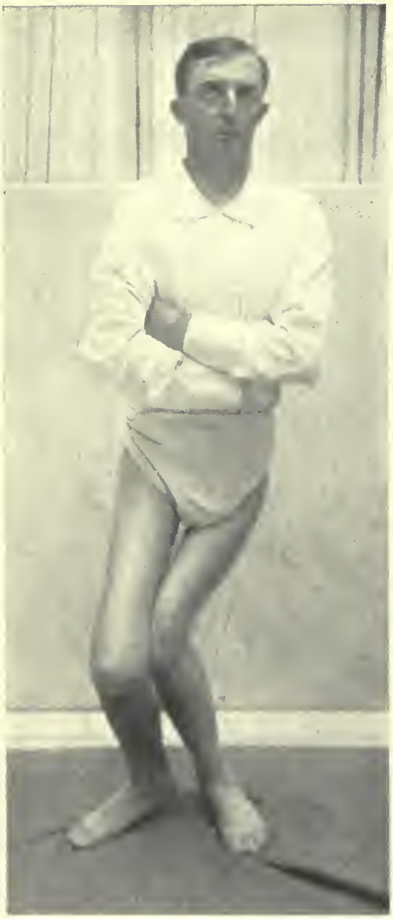
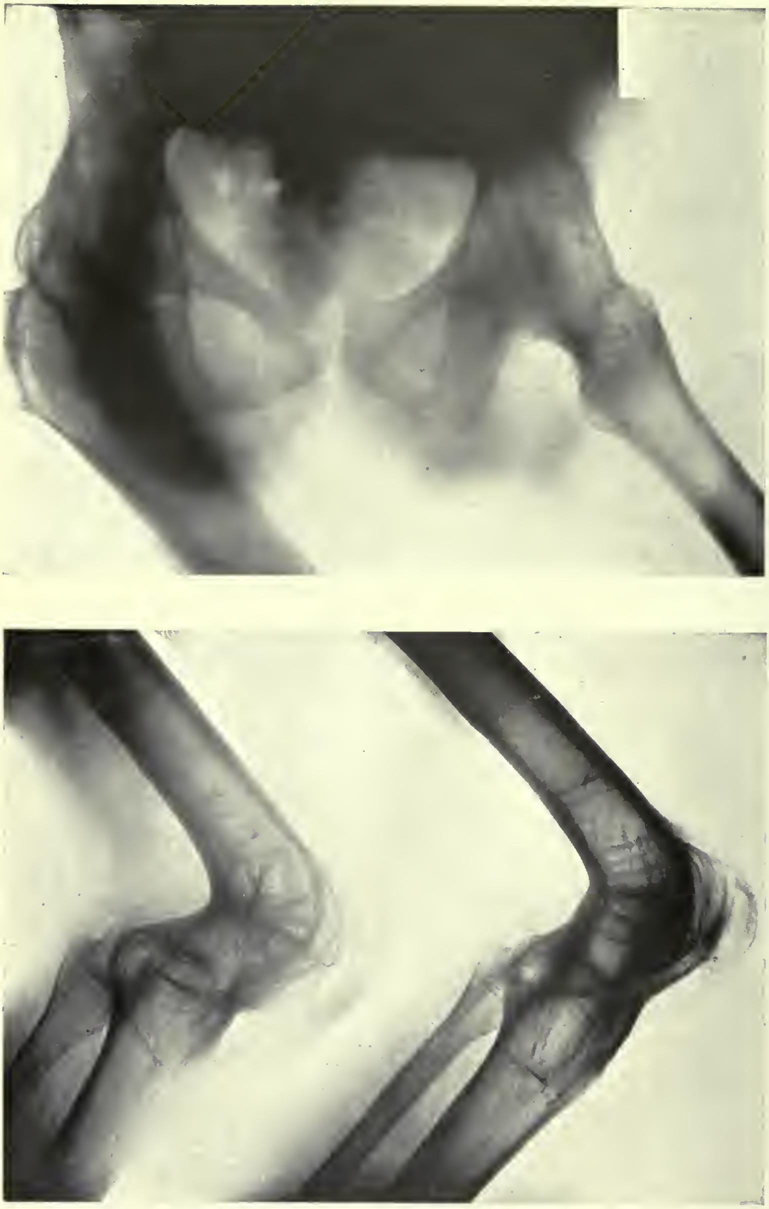


Fig. 31.—Ankylosis of the knee. Showing deformity of legs before operation.



Figs. 32 and 33.—Ankylosis of the knee. Arthroplasty: Fig. 32, Skiagram showing complete bony ankylosis of both hip-joints. Fig. 33, Skiagram showing complete bony ankylosis of both knee-joints.

gained in strength. Fairly well all that summer. In the winter of 1905-06 had great pains on motion in thighs, calves, arms, knees, and hips. Legs were flexed on thighs and thighs on abdomen for first time. At this time knees and hips became ankylosed. In July, 1906, was delirious and had high temperature—does not remember how much; pains relieved some. Went to Des Moines in August and took treatment for the whole winter of 1907, and pains all left gradually, leaving patient in present condition. Bony ankylosis of both hips and both knees with great deformity, as seen in the photograph and skiagrams.

COMMENTS AND OPERATION

This man has already had an operation for ankylosis of the left hip. He had ankylosis of both hips and of both knees. Whether we can make a joint of this knee remains to be demonstrated. This is the hip that was formerly ankylosed. You see the motion he now has. He was operated on ten or twelve weeks ago. This is one of those cases where the man was thrown out of commission for life, so far as usefulness is concerned, and he had nothing to live for unless he could get movable joints—at least in one hip and in one knee. You see the enormous backward luxation. *All that deformity is avoidable, is absolutely unnecessary. The practitioner who treated this case could have avoided much of this trouble by putting on a 20-pounds Buck's extension. He treated this patient for rheumatism, I understand. It is not rheumatism at all, but a metastatic infection. He could have made it an easy matter for us to operate on this patient, instead of making it difficult to do anything at all with the case.* There is no idiopathic acute arthritis: they are all metastatic arthritides.

If you look at the skiagram, you will see there is no line of demarcation—just one solid mass of bone. Here we will put great tension on the vessels. It is a disadvantage to operate on a joint with one already enfeebled, as the upper one is, but we are going to endeavor to do so. I am not going to make my regular flap in this case, as it would be so long that it would die. I am going to make this incision in my flap and throw it out, retaining only the capsule. There is the outer capsule of the former joint. See it! I am going at this on the supposition that I am going to make a new joint, and I am taking my interposing flap from the anterolateral capsule. That is where the interposing flap is made with its base downward. That is the true fibrous capsule of the joint. There is no synovial membrane here. That will be the flap I will interpose between the edges of the bone when I get through. Ordinarily, I would not change the conformation of this femur; I would change the conformation of the tibia, but here I cannot do that, you see, because I cannot take off enough of the tibia to ever get a straight leg, and what I must do is to change the conformation of the femur, which is not changed, as a rule, in my technic. There is the internal lateral ligament which I am endeavoring to preserve to reunite it.

Let the record show that we are preserving a portion of the internal lateral ligament. There is the anterior portion united to the outer capsule with its quadriceps, passing under the quadriceps tendon here, leading down to the ligamentum patellæ. There is my interposing flap. That is the first and important thing. Here is the quadriceps tendon. We must follow that over until I come to the ankylosed patella, and I will split this close up, so that I can throw it out to the opposite side and reunite it. The patella is ankylosed usually over a small area, not larger than a nickel, except in the tuberculous cases. There is the line of ankylosis of the patella. It is not much larger than a nickel. All that end of the bone has softened since it has been out of use. The patella is now free. We will enlarge the incision as much as we need it to allow the patella to be displaced outward. You see the degree of absorption that has taken place. I am leaving this tendon of the patella attached. If this were a straight case and did not have the complications it has, all I would have to do would be to saw off the tibia and swing it round and straighten the leg. I cannot do that in this case. It must be drawn out. There is the line of bony union between the two bones. This makes the tibia conformation right. There is the crucial ligament. You can see it glistening through. Now we are able to form some estimate of about how much more must be taken off to bring the part round, and I find it is quite considerable. I am making my intercondyloid groove so that there will be no lateral luxation subsequently. I am making a semicircle of that. I want to prevent the fat of that capsule from being interposed here. It is sutured now to the posterior capsule of the joint. This is my anterior flap, which we will pull over in the middle in a moment to meet the one of the opposite side, but the one on the opposite side has not been prepared at all yet, but will be prepared now. I am making this flap from the inner side. This is the inner portion of the capsule of the joint, which makes the inner flap. You see the fascia and fat that are interposed. We have all that surface covered. The wire we use is soft wire, and simply covers up the connective tissue. The hazard that will come will be the tension on the nerves and vessels from straightening the leg, but that tension will not be great. I do not hazard this as I did the last case. The last case I hazarded in the superlative degree, but I promise you I won't do it again. In that case I feared the young woman would have a gangrene of the leg. I worried over it terribly; I had prepared her for it, but I had not prepared myself, which is more important for my peace of mind, and when it looked at the end of twenty-four hours as though she would get gangrene of the leg, I felt mighty uncomfortable.

I now interpose the fascia and fat, and all the bone surface is covered. I am throwing the patella back; I am not going to get ankylosis of the patella, because I can luxate it upward materially. I will be able to push it down over the surface of the tibia; all the raw under surface will come down, and I will throw this across here to protect it from adhesion. We will take off the upper portion of the patella. Remember, the patella is an unnecessary bone. The quadriceps tendon is vital to the motion of the joint, but you can take out the patella, as we have demonstrated again and again, and yet have a splendid result by uniting the two tendons across the joint. I am taking off the upper portion to put the patella lower down and shorten the quadriceps tendon that has been overextended by this extreme flexion for this long period of time. I have reduced the size of the patella as much as it should be. Ordinarily, we rotate the patella 180 degrees and put the aponeurosis side toward the joint, but here it is unnecessary to do that, because I am able to come clear across that with my tendon. I show you the tubercle of the ligamentum patellæ. I sew that across here, loosen with chisel, and fasten it down on the under surface of the denuded tibial crest, because we get extra leverage when it comes to sliding over.

Let the records show that we had to take off practically all the patella, leaving just a small portion of the upper end large enough to form a bone attachment to the tibial crest, and we took practically nothing off of the end of the tibia—just enough to make its conformation correct for the new joint; that we endeavored to produce an intercondyloid notch with a fairly rotating round surface, so as to permit rotation subsequently; that the elongation of the quadriceps tendon that had been due to the long hyperflexion was compensated for by plicating the ligamentum patellæ on itself, and putting the patella over the tibia and using it as an elevation so as to give additional leverage in making an extension of the limb; that the interposing flap was entirely an internal flap, and not the semilunar flap we have used heretofore. In this way I feel that the vitality of the flap is very much less hazarded than in the previous operation. There were no ligatures used at all, and by the time the operation was completed there was no oozing; the limb came to a straight position, putting very little tension on the nerves, artery, or vein —very much less than in the last case we operated of this type. An interposing flap was formed in the usual manner. The internal lateral ligament was preserved to a considerable degree and reapplied, so that we have a good internal lateral support. There was no tension on the skin in any position.

Let the records further show that there was abundant skin-flap without tension.

Good results in the knee are more difficult to secure than in any other joint. That is because it has to be mechanically so perfect and plumb in order to get a movable joint with an ample support. That is why I had to round the part off and chisel it off for the support, otherwise there would be pain when the patient walked and the joint would be locked in certain positions. That will round itself off as soon as cartilage forms.

This is the best skin-flap we have had; but we cannot do this in the other type of cases. The skin-flap in the knee has been our greatest source of trouble.

I am delighted with this flap because I feel it is going to live and not need grafting. There is no incision on this side, so that we will get all the collateral circulation for the outer vessels.

We seal this wound up without a drain. This is 5 per cent. carbolic gauze we are using. The reason we use this is because it is material that will take up the oozing and immediately sterilize the secretion. If you use ordinary gauze, where the saprophytes come in they will decompose the secretion and we hope not to remove this dressing for three weeks.

Note.—Primary healing; no flap necrosis. Little or no pain in either hip or knee. Left bed in five weeks.

VOLKMANN'S CONTRACTURE

HISTORY

The patient is a school-boy, aged eleven, admitted because of an atrophy of the right forearm and inability to extend his fingers and wrist.

Present Trouble.—Dates from June 25th, six months ago, when the patient fractured the ulna of the right arm, 2½ inches above the styloid process. This was a simple fracture, and was immediately placed into splints. The arm and wrist were much swollen at the time, and the splint was wound on very tightly. The hand was cold and blue and was very painful. The splint was left on for ten days, and during this period the patient experienced such pain that it seemed necessary to control it with morphin. At the end of ten days the splint was removed and the posterior surface of the wrist was very red and painful, and was the site of several blisters. A cast was put on at this time and left on for two weeks, but the bone had not united sufficiently at that time and the cast was put on and left until August 15th. This was removed, and the patient states that he noticed for the first time that his forearm was smaller than previously; also that his forearm was smaller than it is at present. The pain had left his wrist and forearm sufficiently, so that he attempted to use it seven weeks after the accident, but found that he could not extend his fingers or his wrist.

COMMENTS AND OPERATION

This will be a long and tedious case on which to operate, and it means careful technical work. How long was the hand cold and blue, and how long was it painful?

Interne: For a period of ten days.

Dr. Murphy: The history doesn't say that, does it?

Interne: I do not think it does.

Dr. Murphy: Then do not put it in unless you know it is so. All the mischief in these cases of too tight dressing is done inside of the first three days. That is why I want the record to show that in consonance with the facts. These patients usually have a severe and intense pain, which continues for three days, and then it subsides. The swelling may continue, but the pain disappears because the mischief has been done. That is why it is important to have it in the history. This patient can extend his fingers, but they are contracted. He can extend his fingers as far as they will go, which means that he has no paralysis of the extensor group of muscles and the musculospiral nerve has not been interfered with, and this should be emphasized, because one of the first cases we had of this type, and on which we operated, had all the nerves operated in this region before he presented himself. It is not a nerve lesion; it never is a nerve lesion. It is always a muscle lesion. This is the second case of Volkmann's contracture we have had in one week. It is usually spoken of as a paralysis, but that is the wrong name for it. It is not a paralysis at all. I want the interne to put down on the history sheet "Volkmann's contracture," and not paralysis.

You look at this skiagram, and there you will see what appears to be a very good bone result. There is a displacement of both bones of the forearm according to this other skiagram, and it shows the necessity of making two skiagrams, as one is never sufficient. This point we have accentuated again and again, but people interpret these skiagrams in and out of court in all sorts of erroneous ways, and they get into all sorts of difficulties because they do not recognize the significance of two or three pictures taken at different angles. One should not pass on a bone case on a *single* *x*-ray. You see, there is quite a deformity. That is fully ½ or ¾ inch out of plumb. Wait until you see the arm, as it shows a very much worse condition than the picture itself. If you run a plumb-line through the radius, you will see what it leads to. The line in the lower segment is displaced considerably from a parallel line. When put on parallel lines, the deformity is never bad. You can have a displacement of ½ inch, as in a fracture of the femur, or an inch laterally if you keep them on

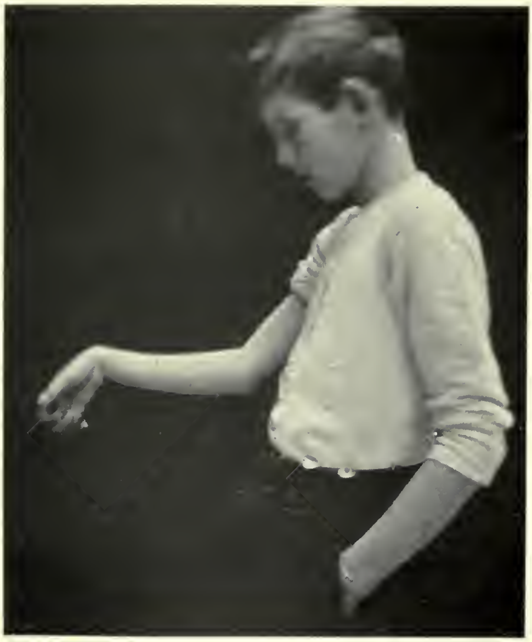


Fig. 34.—Showing case of Volkmann's contracture before operation.

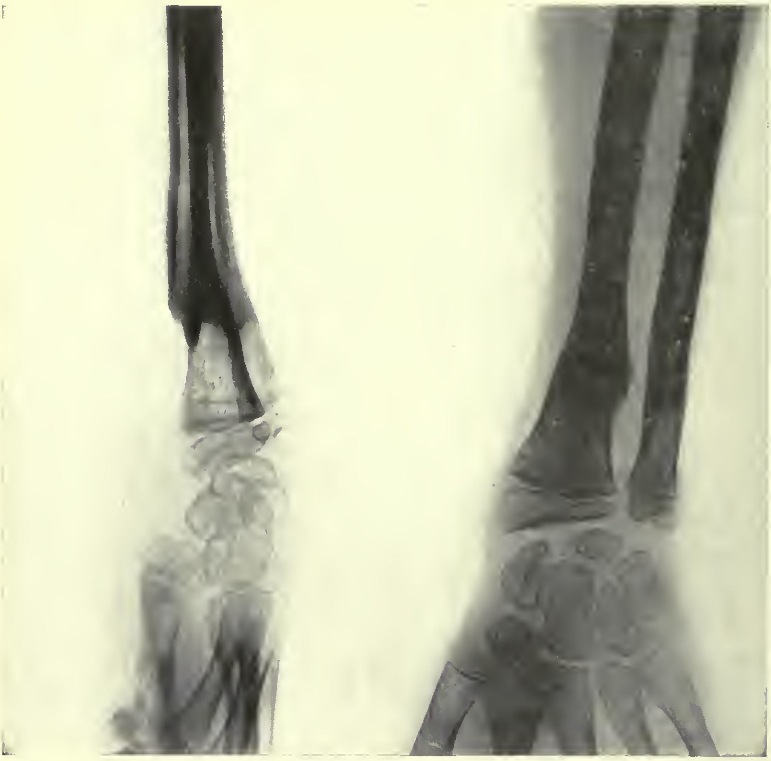


Fig. 35.—Skiagrams showing fracture that was treated with a tight circular case, causing Volkmann's contracture: *a*, Lateral view; *b*, anterior view.

parallel lines. The deformity then is never so bad, but if you change the line from a parallel line, a small deviation gives great disproportion of relationships and pronounced deformity. That is one of the things one must bear in mind, particularly in the femur, and that is why the old-fashioned plan of treating fractures of the femur with Buck's extension has been so very successful, because it has kept them on parallel lines, if not on the same parallel line.

The lesion in this case, and the lesion in a Volkmann's contracture, is a surgeon's lesion, and never a lesion due to the trauma. It is a lesion due to a *defective dressing*, not due to an injury at all, and the difficulty in this case was that the doctor, after using a good deal of, but insufficient, force in endeavoring to correct it, put on a firm binder to try to force the bones in position, which is a mistake. If the bones of the arm or forearm are put in apposition, they do not need a firm binder to hold them. If they are not put in apposition, a binder does not bring them into apposition.

Now, in a Volkmann's contracture you have an apparent paralysis occurring in two positions—the arm or forearm. First, it is most commonly associated with fractures of the elbow; second, with fractures of the forearm, and, third, with fractures of the upper arm.

There are three sets of muscles involved when a fracture occurs in the elbow-joint. You may or may not have an ankylosis resulting from the fracture, depending upon the period of time that you keep it wrapped up in this excessively tight bandage or binder and the degree of trauma to the joint. What is the lesion in the arm or forearm due to? To the excessive pressure produced by the bandage during the stage of acute edema and swelling following the trauma. It produces what? It produces not a neuritis with paralysis: it produces a myositis, and it is one of quick formation. You have ischemia and trauma of the muscles from the swelling; then comes the fibrous contraction of the connective tissue around the dead muscle-cells, and that is what brings this arm into that drawn position. When only the forearm has been compressed, as was true in this case, then you have only contraction of the forearm muscles with flexion of the hand and fingers.

In this other boy, who had a fracture of the elbow with so perfect a result, you can scarcely see that there had been a fracture. It is difficult with the skiagram to see that he had ever had a fracture. He could flex it a little and move his fingers down here [indicating], but contraction pulled his fingers into deformed position and gave him a worthless arm and hand, with contraction of his biceps and brachialis anticus from the upper arm, drawing his forearm into a flexed position and fixing it there.

In the management of these cases, where the muscles and joint are involved, you have the question, first, of arthroplasty to remove and cure the ankylosis; second, you have the question of elongation of the tendons in the upper arm, to permit the extension of the forearm, and muscle and tendon elongation is not sufficient. The all-important thing in preventing extension of the forearm after fixation for a long time, even without ankylosis, is the capsule of the joint. In other words, anterior capsulotomy must be done at the elbow; merely elongating the tendons of the biceps and dividing the fibers of the brachialis anticus is not sufficient always, as we found in the Kilpatrick case, a boy eleven years of age. There was flexion of the hand. He could not get it beyond this position [indicating by photograph], and he could do nothing with his fingers. We all agreed that if we could divide and elongate the tendon of the biceps and brachialis anticus it would come down. After we elongated it, it went down ½ inch more than before, showing that while the primary muscular contraction was an important factor it was not the only factor, as after eleven years the capsule of the joint had contracted. In fracturing the capsule of the joint, after the trouble has been present a long time, one is more likely to break the bone than the capsule. It should be divided with the scalpel.

We must do an operation in this case for elongation of the tendons. The moment we try to straighten the fingers, see what happens! It is impossible to straighten them more than this [indicating], showing the great contraction that has taken place in the tendons in six months. We must elongate every tendon just above the wrist. He has left a little muscle in the middle of the forearm, and all of this must be preserved.

Let us return to the Kilpatrick case for a moment. In that case we cut down onto the internal condyle of the humerus and onto the external condyle, and followed up the condyloid ridge on both sides, and lifted from the humerus and anterior surface of the joint all the muscles, arteries, and nerves, so that I could look right through across his humerus, just above the articulation, from one side to the other, leaving the periosteum and capsule intact. Then I took a tenotome and divided the capsule right straight across and the forearm fell down. I did not have to push it down; it fell down straight, because it was the capsule that was retaining it after the muscles had been elongated.

Capsulotomy in the future must be considered one of the essential elements in the management of articular contractions. It has been overlooked. The orthopedists have endeavored to produce its fracture by the force they have exercised, and in doing so have traumatized the joint when they have not fractured the bone. This is entirely unnecessary; it is unsurgical and inartistic.

Asepsis is here again the all-important matter. If we do not get it, we are worse off than before. We do not have to divide here the anterior capsule of the wrist-joint. I am going to make my incision higher up than in the case of Kilpatrick. Every finger touch means a touch of danger. These tendons have to be elongated to various degrees, depending upon the particular muscles, but here they will run 1½ to 2½ inches in the elongation. In doing this elongation we take each of the tendons separately. There is the median nerve. You can always tell a nerve from a tendon, as it has an entirely different appearance. There is the median nerve, and there is the tendon. This tendon has to be divided and elongated. I split it first. I will elongate it 1½ inches. I overlap that as much as will be necessary later, just fixing both ends on the same thread, so that we do not connect them wrong later.

Now we are going over to the ulnar side. There is the flexor of the little finger. See it move! Here is the flexor of the next finger. Next comes the ulnaris, which is the farthest out of this group, and with that comes the ulnar nerve, and that is the most important nerve from here down. The ulnar nerve supplies all the small flexors to the fingers except two, and supplies three muscles of the thumb, so that you have in the ulnar the most important nerve in the palm of the hand. There is the flexor ulnaris. This tendon does not need so great a degree of elongation as the other tendons, because it has a much shorter range of motion. This will be elongated here only about an inch. There is the artery and there is the nerve. We are coming back on the deeper group. We are down to the superficial ones, sublimis digitorum and the profound group are the deeper ones, and they commence to show up at once in the dissection. We carefully divide the tendon capsule. They come down as a common tendon. They are side by side. I am not going to make the bone operation now. There are the outer sets commencing to contract. This tendon is brought out in the other direction, so that we just elongate it on the flap. This one must be elongated to the full extent. This is the profound group, and there you have to span the longest distance you can get in this incision. Here is another member of the profound group controlling the little finger. Now we are getting them well divided and grouped. This one we will elongate with the catgut as a guide, on the same principle that one elongates the tendo Achillis, but no other tendon reproduces without a guide as extensively as the tendo Achillis.

I am back again on the median nerve, which I demonstrate with the faradic current. We will go over to the radial side. Here is the palmaris longus. I was looking for it, but it did not show up in the right position, and that is because I did not make my incision to the ulnar side. It is a small one, and goes to the tensor fascia of the hand. It does not go to the fingers at all. It does not need the great elongation that the flexor tendons of the fingers need. It is very thin and ribbon like, but we will be able to unite it by putting a loop stitch through. This is the carpal flexor you see. Here is the ulnar. Now, some of these will be elongated more than is necessary. I expect to have none of them elongated less than is necessary. If you elongate them too much, the muscle readily adapts itself to them. If you do not elongate them enough, you have a recurrence of the condition you had at the time of operation. This is why all this work must be done so cautiously.

Now I am getting over into the pollicis group, as you see. I draw these tendons down through the orifice just as far as they will go. I am stretching the capsule of the finger-joints so that they come around. I do not have to break them, because it is such a short time since the accident occurred, and we do not have to fracture the capsule. They are all elongated, and we have corrected the fixations that have occurred in the capsules. Now we are ready to make our connections. There is not the slightest tension on this chromicized catgut suture when we are ready to tie it. Here is one of the greatest elongations; it is just all it can do to touch. I put catgut through the middle each time, and half of it is contacted, so it is a side approximation with that knot. This is the heavier tendon, and will need more than one stitch. With the smaller ones we can do with one. This must be 1¼ inches of elongation, and still it is taxed to its maximum. I am endeavoring to have no two of the unions on the same level, and that is important so that the ends do not cross in their adhesions. If you have them on the same level, there is danger of crossing of the adhesions and fixation of all the tendons in one group, so that they would move synchronously in place of separately. Here is the middle finger tendon split which I cannot approximate at all and will not try. I will lock that at a certain distance and have a guide to its reattachment.

You may ask the question, Why do you not correct the bone deformity at this time? There are two reasons for this: First, I would make an additional wound here and open the bony surfaces to infection where I have done so much dissection as I have here, and would run the risk of getting the tissues reduced below their resistance and having a suppuration. Second, I would have a compound fracture at the position where the tendons were divided, and get a fixation of all tendons in that cicatricial mass to the bone, and not only nullify this result, but spoil it for the final condition, which I could never subsequently correct. I have now elongated all these tendons.

The next and one of the most important steps is to cover these tendons up separately with a sheath so that they do not agglutinate. There you can see at once is the radial group. I will shut this off by taking this loose tissue—this loose web of tissue here—and anchoring it. There is some fat; I will pull that in and hold it there. I am making new sheaths for the tendons, or compartments for them. Now we will place the ulnar group into compartments. The ulnar nerve comes in here [indicating]; we can take its sheath, and in that way shut off the flexor ulnaris at once without disturbing either the tendon or the nerve. We shut it off: it is all ready in its compartment. I have every one of the tendons separated as far as they can be. I will commence and unite the skin with the subcutaneous stitch, so that the tendons will not become adherent or fixed to the skin. I will continue this suture so as to make a complete separation of the skin and tendons in every position, and then we will get a separate gliding of them.

Let the record show that on the completion of this operation we were able to make a good elongation of all the tendons, to get separate unions at points distal, with two exceptions, and to secure at the completion of the operation a complete extension of the fingers without external support. I gave you only one of the means of overcoming this shortening of tendons—that if this contraction, which is only six months old, should continue, with these tendons not sufficiently elongated, then I may resort to the second means, which we did in the case of an automobile accident a short time ago, namely, shorten the bones of the forearm. In making that procedure we availed ourselves then of a plan which was originally suggested for this condition, namely, taking out a segment of bone and shortening the bone so as to permit the tendons to functionate properly. That, in our opinion, is not the proper operation. The best operation is the one that we have performed. It took, as you saw, a considerable period of time for its performance—nearly two hours.

In the case I cited—in the automobile accident case—the barbed wire tore out something like ¾ inch of all extensor tendon groups and muscle groups at the junction of the tendons with the muscles. There we shortened the bone about ¾ inch. In the first place there was non-union and part of the bone was gone; in the second place it gave us an opportunity to get tendon apposition across the line of bony union. In that case we were able to cover all the bony surface with a fascia flap, separating it from the tendon, so that we expected and believed we would have, as we did, primary union of the soft parts, and we had union without fixation of the tendon or muscles to the posterior surface of the bone. But there is no occasion, in the case before us, for taking any such chances and the bone is not disturbed. We dress it in a straight position. There is no tension or pressure over the point of union so as to force the tendons into each other. We give them all the play we possibly can.

What will be the final result in such cases? Where you have a sufficient quantity of muscle-fibers either above or below to put the tendon through the necessary contraction, you get perfect restoration of function.

Dr. Rexwald Brown was an interne in this hospital and suffered from a Volkmann's contracture for years. In his case the nerves were operated before he came. The condition in his case was that he had enough muscle belly left so that when we straightened and elongated his tendons he had sufficient contraction to put it through every range of motion. He has an absolutely perfect hand.

In another case, a picture of which we showed you, the one that had the upper and lower arm contraction, he has a perfect hand and a perfect forearm. He has perfect extension and flexion, and can touch his finger against his shoulder, and has every use of each and every finger. He is a good piano-player, showing how perfectly each of the tendons can move separately when you take the precaution in the operation to insert that little mass of areolar tissue between them.

We have a case from Seattle which is not going to give us as good a result. The patient has a serviceable hand. Each of the fingers moves separately and individually, but he has not a wide range of motion, because he has just a few muscle-cells left in his long flexors and, in addition, his median nerve was operated and has resulted in a paralysis of the long flexors, the greatest portion of his compression having been produced on the anterior side of the forearm [indicating]. In addition to that the boy from Seattle has had three operations on the elbow-joint for ankylosis, two of which were done before I operated on him, resulting in a return of the ankylosis, and one by me, with the interposition of fascia and fat, with success as far as bony union is concerned, but with too great limitation of motion. He has a large cicatricial bony mass in front from the previous operation on the elbow. To dissect that out we are going to take out a portion of the coronoid process that presses up into the fossa of the humerus, that has been filled up with an exudate or callus following the fracture. We are also going to take off a portion of the tip of the olecranon, but as he is still growing, we dare not invade the epiphyseal line on either bone.

I will show you in this connection the picture of a case in which we had a failure, as far as sufficient motion was concerned in the early stage, and then we did a minor second operation that laid the patient up for ten days for union of the soft parts, and she has perfect restoration of function by doing the operation which I have just described, taking off the tip of the process and olecranon. Here was the original operation. When we did this operation we regularly divided the olecranon, but we are not doing that in the modern procedure. We stretched the wire which fixed it to the ulna, and got a bony union that left a V-shaped articulation. In the next operation we took off the tip up to the point I show you. These pictures show the extent of her extension and flexion. There is full and complete restoration of function. She has all the motions of both the forearm and her elbow. Pronation and supination are perfect. She can do anything with that arm that she can do with the other arm. This is what we shall do with the boy from Seattle, so far as motion in the elbow is concerned. We shall not be able to do anything further with his tendons because he has not enough muscle to get full range.

ANKYLOSIS OF THE HIP—ARTHROPLASTY

HISTORY

Patient is a female, by occupation a cashier, aged thirty-eight.

Present Trouble.—Started in August, 1909. Patient had a severe pain in the left hip. She was walking on the street at the time and it caused her to stop—the pain went away in a few minutes and she walked home and continued about until the third night, with only slight twinges of pain. She had no temperature, no cold, no tonsillitis, or gastro-intestinal symptoms at that time. On the third night following she took a very hot bath, retired, and woke at two in the morning with an excruciating pain in the left hip, which radiated down the limb to the knee and ankle. Fifteen minutes later she had a severe chill and began to feel feverish. Her pain was so severe that it was necessary to control it with morphin. The temperature was 103° F. that night, and the next afternoon was 104° F. The patient remained in bed and was very ill; the limb was so painful that it could not be moved, and she was taken to the hospital and an extension was put on for fourteen days. The patient was confined to bed for three months. The pain subsided somewhat and took the form of a dull ache, but the hip was extremely painful on pressure or movement. The patient lost 55 pounds in the three months and ran a temperature for two weeks.

At the end of the second month in the hospital she began to sit up in bed, and then noticed for the first time that there was a limitation of the movement of the hip. She was unable to flex the thigh upon the abdomen or to abduct the limb to its maximum. She left the hospital in three months, but was unable to walk or bear her weight on the limb on account of the weakness and soreness in the hip. Six months after the first onset of the trouble she began walking with a cane. The patient walked without the aid of a cane eight months after the onset of trouble.

A year and a half after the first onset of pain she was taken to the hospital, given an anesthetic, and the limb was forcibly flexed, and an endeavor made to overcome the trouble by stretching. The patient noticed an increased motion of the hip-joint after this. At the present time she is not able to flex the thigh on the abdomen except as she flexes her pelvis. There is a complete limitation of abduction and adduction. All movement in the diseased hip comes from movement of the pelvis; absolute ankylosis exists in joint.

COMMENTS AND OPERATION

Dr. Murphy: You say the pain started in August?

Interne: Yes.

Dr. Murphy: What day?

Interne: In the middle of August. It was on Tuesday.

Dr. Murphy: All right, Tuesday. Put down Tuesday. Why do we want to know whether it was Tuesday or Friday? Because the trouble began in such a stormy way as to impress permanently upon the patient's mind the time of its occurrence. That is why. We do not care particularly whether it be Tuesday or Friday, but we do care about interpreting the severity of the initial symptoms. If we carefully analyze the points in the history, we can form some estimate as to how severe the primary attack of this disease was, and this is important in considering the diagnosis made at the time and the pathologic conditions that subsequently developed. What year?

Interne: 1909.

Dr. Murphy: That is correct. What was the original diagnosis?

Interne: I do not know.

Dr. Murphy: I want to show you, if I can, the reason why the diagnosis which was made was erroneous, and why one should not be misguided into that diagnosis. This patient had severe pains which were called sciatica. The pain was in the hip, and called sciatica, which does not begin with severe pain on slight movement of any kind. In sciatica it is painful when you put tension on the nerve, but it is not painful with the slight movements when you do not put tension on the nerve, differing greatly from the lesion from which she suffers and which gives pain on the slightest motion of any kind in the joint. She has slight motion of one or two degrees, so that it is really questionable whether there is any free motion at all in the hip—that all the flexion is a pelvic flexion. In a careful examination I made yesterday I could not determine any motion in the hip-joint free from the motion of the pelvis, which she has cultivated to so great a degree on account of fixation, as in any case of bony ankylosis.

Interne: There are only two or three degrees of flexion motion in the joint.

Dr. Murphy: It is questionable whether there is any motion at all in the joint. There is the margin of the acetabulum around the neck, out to and almost reaching the trochanter. This is, we believe, a self-limited lesion. We think there is just the slightest rotation of the head; we believe that it is not a bony ankylosis of the surface of the acetabulum, but that the condition is due to bone developing from its margin out over the neck, following the firm connective tissue which forms the outer layer of the capsule, and which, in the embryo, is a continuation of the periosteum and a great factor in the genesis of bone, the tract that ossifies and gives us the bony system.

In the man I operated on a short time ago who had a spirochetic infection you remember how beautifully we were able to demonstrate the projection of bone in the hip extending not only beyond the embryologic limit, but extending in identically the same embryologic tissue to many inches beyond the anatomic limit.

In this case it looks as though there is a line all the way around, but remember this trouble is of some years' duration, and there should not be any cartilaginous line at all if it is a bony ankylosis. This bone has gone clear out to the trochanter. You see here where it impinges apparently, where there was suppuration or caseous degeneration and possibly necrosis of bone, and there it generated another mass on the under surface of the neck.

This is a splendid skiagram showing the condition (Fig. 36), and without this class of pictures you are absolutely at sea. No one can do bone work successfully without the use of the x-ray, because without the x-ray to confirm it, as in this case, I might start out with the proposition that this was a bony ankylosis throughout, but with the x-ray picture we find that there are other reasons for the inhibition of motion through the mass of callus that is outside, this differing entirely from the mass of callus we had in the last case. You remember in the last case we had motion was limited, due to the fact that he had no bony ankylosis of the head, but following the fracture there was enough cover of bone all around that would impinge everywhere. The patient was a miner.

I am preparing to do an arthroplasty. It looks to me as though the limit of motion is not due to bony ankylosis of the acetabulum, but to a mass of bone developed from the margin of the acetabulum and surrounding the neck, and inhibiting motion in that way.

The history is beautiful. *A history should be a citation of facts in the order of their occurrence.* She had first a little pain three days before. It caused her to hesitate, to stop on the street. The third night after that she took a hot bath, and in the middle of the night was seized with a severe pain; then came a chill, followed by elevation of temperature, with all the evidences of a metastatic arthritis. Then came immobilization immediately because it gave such great pain. The cartilage withstood the onslaught; the capsule did not. The cartilage apparently in this case is preserved, and has not been destroyed by the infection. Then came the long period of invalidism, with only *two* weeks' extension, when it should have been *five or six* weeks' extension, with an injection into the joint to stop the inflammatory process, but, above all, preventing adhesions in the joint until the inflammatory symptoms had subsided. One fortunate thing about this case is that extension has been applied

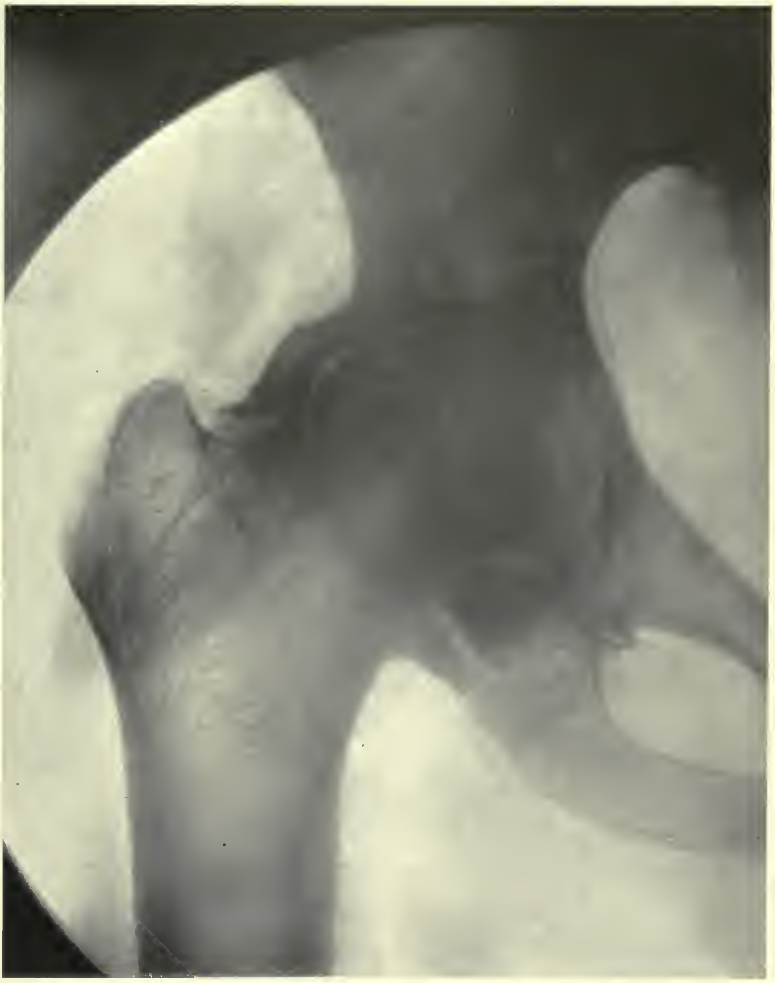


Fig. 36.—Skiagram of case of ankylosis of hip, showing complete ankylosis with collar of bone on top.

and very admirably, having kept the limb in a perfectly straight position all the time. If you have a sufficient weight on, say, 20 to 30 pounds, you will always keep the limb in a perfect position during the process of inflammation, and you will get rid of that deformity, even if it ankylosed it in the best possible position for usefulness of the limb subsequently, making so great a contrast to the case that we operated here on Wednesday. If you remember, we had a man here with the legs at an acute angle with the thighs and his thighs displaced laterally to his pelvis and all four joints ankylosed. That man has not had an unpleasant symptom. We operated on the knee, after having previously done an operation for ankylosis of the hip.

There is no doubt but that we shall be able to get a strong, serviceable limb for this woman, although the leg has been out of commission so long. You do not need to give these patients morphin or anything else in the acute inflammatory stage of the disease if you put on extension sufficient to prevent the contacting of the inflamed surfaces, which means *adequate* extension. What you want to do is to relieve the patient of pain by extension, *and extension does relieve pain.*

Here is a case [showing picture] which shows bad management of a metastatic infection of the knee, with the knee ankylosed at the angle you see. This is all avoidable by the doctor putting on, the first day of the rheumatism or metastatic infection,—call it what you will,—simply Buck's extension.

In this case we have probably a bony ankylosis, but if you were to ask me to interpret the condition, I should say that it is not a bony ankylosis of the acetabulum.

You see the trochanter. You will notice that there is some displacement upward. There is some absorption upward. If you take and compare the position of the lesser trochanter, which you can see coming out, it is fairly well up to the level of the tuberosity of the ischium; there is a great thickening around the neck which I believe is mostly due to the callus formation. That will give us difficulty is using the chain-saw to remove the trochanter. When you look at the picture you will see that the neck is shortened, because the callus has filled it all out and you do not see a clear neck. I am beveling this portion under because I want to take more of the fascia lata than I do of the skin. Our skin-flaps have been our greatest trouble in all our arthroplasties. I am now dividing the fascia lata. I am taking a larger flap of that than I am of the skin, because I need it to cover the head. There is the trochanter [indicating]. Here is the gluteus muscle attached at this point. I will prepare the posterior surface of the trochanter. Remember, in going around on the posterior surface we have three important muscles; following the line of cleavage you see how nicely you come down to the situation. You commence to get on the margin of the gluteus medius, and then, going in here at the side, we have the attachment of the pyriformis, and the two obturators going in to be attached to the under surface—base of trochanter. The next step is to get off the trochanter. When the trochanter is off, then we are mighty close to our field of work. We do not cut the muscles attached to the trochanter at all. There is the chain-saw carrier. I have just gotten inside it right. The other is below and must be released. I want to get under the other muscle and suture it to the one that is still attached. I put a suture in now, so that I won't have to look for it afterward. I remember once, when working in a callus, I thought I had cut the sciatic nerve. I did not get under the muscle with the chain-saw, as I had hoped, but I am fixing it to the position in which it belongs, right at the base, with the one that is intact. We have been using phospho-bronze wire, but I am not going to use that suture any longer close to the skin because it occasionally is the source of irritation to the patient. Patients tell me that they feel a pricking from the wire like the pricking of a needle.

The next step now is to get in onto the articulation. Now I am in on the neck. I want you to notice this great callus. It shows beautifully. I may cut the capsule right through and make a dissection of it. It goes away down, as shown in the *x*-ray, clear out to the neck. There is the trochanter minor. Here is the margin of bone I want to call your attention to again, coming right up to and limiting motion. We stop all this bleed-

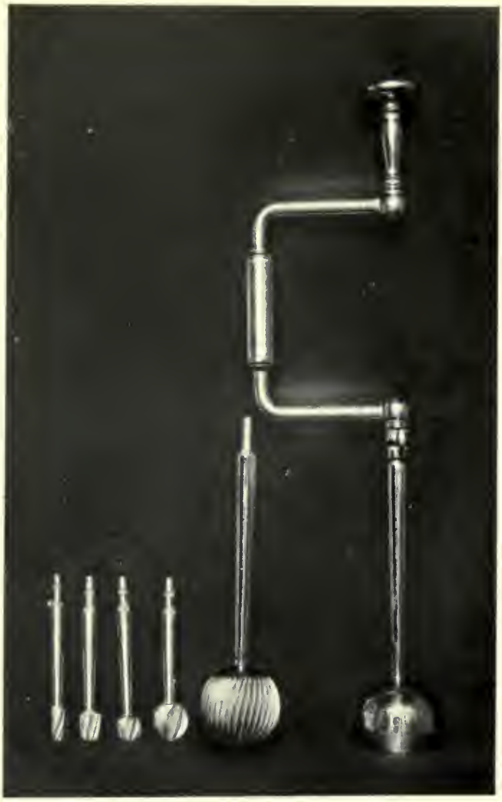


Fig. 37.—Rongeur and burrs and neck conformer used by Dr. Murphy in arthroplasty of hip-joint.

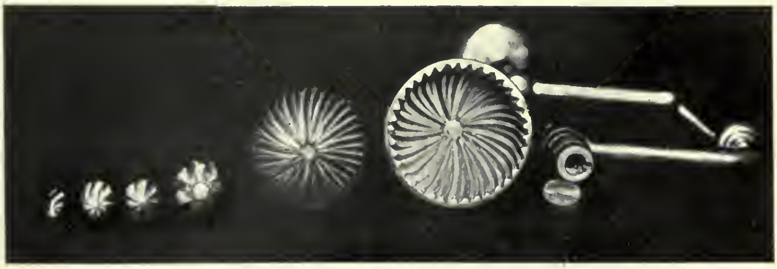


Fig. 38.—View showing inside of neck conformer and end of burrs.

ing with the Blunk forceps. It is a good instrument for moderately sized vessels that are deeply situated. I have my field prepared, and I am ready to go to work with the chisel and take off this margin of bone. There is a large collar of new bone that has been produced around the acetabulum and is shown exactly in the picture.

I have to do an arthroplasty in this case. This is a much more difficult operation than a regular arthroplasty. The picture shows that there is no bony union. Let the records show that the picture is still correct; that the lower half of the bone is ankylosed at one point, but the major portion of the head is not.

Now we are ready to change the conformation of the acetabulum to enlarge it sufficiently to accommodate the head and flap. The bone work is done. It was a more difficult task than doing an operation with no motion at all—very much more so; at the same time it works out nicely. It clears up nicely the conditions. Now we are getting the flap ready. Whether this flap will continue to live I do not know, but that does not matter very much, so far as we are concerned, so long as it does the work. I will cut off the two muscles again, keeping them both to go over my trochanter and put them in beneath the muscles. Now we have the flap ready to fix around the neck.

Let the record show that the acetabulum is perfectly clear; that the skiagram showed that there was a cartilaginous mass between the approximated surface of the bone, and that is true, but it was difficult when we were inside to discern that there was any motion, and we had, to all intents and purposes, to deal with the case as one of complete bony ankylosis, as we had to take off all the cartilage that remained of the acetabulum and much of that of the lining of the femur; that the elongated bony margin of the acetabulum was all chiseled away down to the normal level of the acetabulum, as near as we could estimate.

We are fortunate in having two cases of advanced repair of this kind in the house at present. One patient came back on account of suppuration that formed around the nail in the trochanter. Unfortunately, in that case I cut off the head of the nail. It is the only case in which we have had a suspicion of trouble from the nail, and it is one in which it is going to be a difficult task to get the nail out if it is the cause. The patient has a splendid result, aside from the recurrent attacks of infection which involve the cellular tissue outside of the trochanter, and not the joint at all.

The fascia lata is now sutured to the capsule around the acetabulum so loosely that it indents sufficiently to allow the head to carry it deep in the cavity now when I swing the thigh around and replace the head.

We first took off the margin of the bone until we got down to the normal depth of the acetabulum; then threw the head of the femur out of the acetabulum, and in throwing it out we fractured off a portion of the under surface of the head, leaving that adherent to the acetabulum. This was taken out with the rongeur and with a chisel so as to clean the acetabulum fully; then the acetabulum was enlarged amply for the reinsertion of the head with the flap. When this was accomplished, the flap was prepared and sewed in. The head came over in this way [indicating], pressing it in. The process was longer than the usual process, because we believed we had cartilage that could be of service, and that is why we took off the margin before luxating it. We did not put on any extension. It will be dressed in the abducted position, like a case of fracture of the thigh. The extension will be put on in twenty hours.

In the final results of these cases we have been very much gratified that we have not had subsequently bony ankylosis in a single hip—gratified that we have had ample motion in every one of the cases from the first. It is very nearly ten years since we did the original operation, and we have carried out every single step today that we did then except taking off the margin of the acetabulum before luxating the head. We use no drainage and no ligatures. We rarely ever use a ligature, as the blunt forceps stops all the bleeding that amounts to anything.

Here are illustrations of a case of bony ankylosis from a bullet wound. The patient, a boy, had a bullet wound through the pelvis, fracturing off the head of the femur, and subsequently I took the head out through the pelvis. I went in through the pelvis, chiseled out an opening on the same side of the pelvis to allow me to take the dead head out through it, and took the head out through the upper part of the pelvis, because the femur was ankylosed at the margin. I followed the sinus from the bullet wound and took out the dead head. He had complete ankylosis, so that the leg went out at right angles with a plumb-line through his body. The bullet fractured the head and infected the acetabulum. He had suppuration of the acetabulum around the dead head. When that was healed, six months later I did an arthroplasty successfully.

In these cases I usually take a big curved chisel, go down to the depth of the normal head in the ilium, go clear round and fracture it out, as we did in this case, enlarge the acetabulum, round off the head, put it back in, and nail the trochanter back. This operation was done nine years ago. I will pass around some photographs showing the degree of extension and flexion which this young man has. I demonstrated this case at Seattle last August in returning from the meeting of the American Medical Association. He has perfect motion in that leg. He is on a ranch, and can ride ten hours a day on horseback. He has his abduction, which is the all-important thing. The limb is short; as the osteogenetic epiphyseal line was broken off by the bullet and infected, it was removed, and the boy was only about nine years old at that time, so his femur did not grow in length from the time of accident.

PROLAPSUS RECTI

HISTORY

PATIENT is a farmer, aged forty, who entered the hospital December 19, 1911, on account of a prolapse of his rectum. Family history negative. Has never been ill with the exception of the present trouble. Uses tea and coffee in moderation, but no alcohol. Smokes two cigars a day. Venereal negative.

Present Trouble.—At age of eight the patient states that a small portion of the rectum protruded when he went to stool; this could be easily reduced, but has continued to prolapse more and more, and the prolapse is sometimes rather difficult to reduce. In December, 1909, the patient states that he had a slight hemorrhage while at stool. Since then he has had three small hemorrhages while at stool. The patient states that this prolapse has caused him no pain at any time. He has been constipated all his life. The protrusion is now as large as a man's fist.

COMMENTS AND OPERATION

One of the most disagreeable conditions to deal with is a prolapse of the rectum that has reached the third stage. Patients of this class have generally been cut, sutured, and cauterized, and all such efforts have been characterized by failure. These cases of prolapsus recti have been fixed below and fixed above, and the ultimate result has been many failures. We tried to fix the prolapsed rectum above by a plan we outlined some years ago, and it was successful in the cases we had at that time, but we are happy to say that we have very few such cases. We are also sorry to say that we have more than we desire.

Fixation in the abdomen, we believe, gives the best results in the superlative cases.

I am going in here, as you see, in the outer portion of the left rectus muscle at the linea semilunaris. You see it shining through there. I wish to make a definite plan of fixation in this case, but it will depend largely on what I find. We will take the inner layer and displace the rectus muscle inward. Here is the second layer. You see how the second layer retracts outward. This must be fixed before we start to do any other work. If we do not, we are likely to have great weakening of the abdominal wall. There is a little tension here. When you open the abdomen on that plan you have air rush in, but this patient is under forced inspiration on account of laryngeal obstruction, and the intestine slips out in the field. As soon as the patient is fully under the anesthetic we will pick up the sigmoid and make an inspection and see what will be necessary to do. Here is the sigmoid. It is now in position. It has a long mesentery. I want to show you the alteration in the upper portion of the sigmoid—that portion that has been depressed. It folds everywhere. You see the surplus of it. You can take it out and run around the corner with it, which accounts in part for the situation. Here it commences to be thickened. At one place above it is narrow, and in another below it is large and folds down.

I want to do this operation the same as I would do a pelvic operation on the female. Now I have a good exposure. I must draw the sigmoid up and up. This is a place where the small intestine is forced down into my field, and I do not wish to have it there. Here is the cecum, which is coming down to the rectum. There is no tumor here. The mesentery of the lower sigmoid is short toward the middle side, and is displaced downward quite a little. We will commence and shorten that and implant the sigmoid under the parietal peritoneum all the way up here over the iliac artery. There is the iliac vein, and here is the ureter at this point [indicating], passing under my finger. You see the deep epigastric. I am going to implant the sigmoid in the connective tissue beneath the peritoneum in the wall of the abdomen as far to the left as possible and up to the level of the umbilicus. If you divide the peritoneum and elevate the external flap for a distance of eight inches, you can then marsupialize the sigmoid beneath the peritoneal flap and suture it firmly to the abdominal wall muscle: nature will do the rest.

The redundancy of this is seen here. I show you the vas deferens and seminal vesicles coming up from the patient's pelvis. These are the epiploic attachments to the sigmoid. I have gone in behind the peritoneum and have made a pocket for the sigmoid, and I am going to implant the sigmoid behind the peritoneum. All the peritoneal portion of the intestine will be extraperitoneal. I am sewing this to the lower margin of the split and implanting it extraperitoneally; then I am going to get this flap so that I can put the peritoneum in such a way as to adhere to the connective tissue. This is the plan we outlined years ago, and, as we have previously said, was successful at that time. We are using ordinary catgut. It does not matter what catgut you use, as the final result is not dependent on the catgut. All these epiploic tags are fixed extraperitoneally inside my posterior suture line.

Having the inside bowel fixed, I am now going to the upper surface of it, and fixing the external peritoneal flap over the bowel clean down to its mesenteric border, so the bowel is completely out of view from the peritoneal side. This is an extraperitoneal implantation of the bowel, so that there will be as little pressure on it as possible in future defecation. I am working now in one of the dangerous zones for infection—in the subperitoneal connective tissue. It is one of the great danger zones; it is one of the places where we are very likely to have infection in all our experimental operations on dogs. To prevent any possible pressure I have the bowel fixed, and it is extraperitoneal. These epiploic tags have all been buried in. Our plan is to implant the sigmoid outside of the peritoneum and never leave any loose sigmoid in the peritoneum.

Let the record show that this patient has no neoplasm of the sigmoid, notwithstanding he had some pain a short time ago in his left side. Now we close this ectropion. We hope, when the sigmoid becomes secure in that position, as we know it will, not depending upon the sutures, but upon the adhesions which will form between the peritoneum on the dorsum of the sigmoid and the connective tissue behind the peritoneum, it will go back to its normal contracted size. If the patient should have some exfoliation of the anus, which he might have, we cut that off and cauterize again, but we probably will not have to do this. The anal fold, which for years was ectropic, may come out when we have made the fixation. We fix for six or eight inches the area that has been loose for the folding down in defecation. This is closed in the regular way,—the ectropion of the serosa,—so as not to get adhesions to the anterior abdominal wall.

Note.—Primary union. Bowels move without the aid of cathartics and without anal ectropion.

EXPLORATORY LAPAROTOMY, APPENDECTOMY, MEGADUODENUM

HISTORY

MALE; age, thirty-six. Admitted December 18, 1911. Has been married sixteen years. Uses no alcohol. Family history negative. Has never been ill before the present trouble. Venereal negative.

Present Trouble.—Trouble started four years ago, and has been continuous ever since. Pain begins from one to two hours after eating, and lasts until the next meal, when eating relieves it for from one to two hours. Pain is in the epigastric region and does not radiate. Liquid foods give less distress than solid. Alkaline solutions relieve pain more than any other treatment. He wakes up at 1 or 2 A. M. with the pain, when a glass of water gives him relief. He is nauseated after each meal, but never vomits. Has never vomited blood. He is tender over an area the size of a dollar to the right of the median line, just below the costal margin. Stools dark in color; has been taking medicine, but does not know whether bismuth is in it or not. No bright blood. For the last two months has been having fainting spells about every fourth day, but does not know the relation of the pain preceding these fainting spells or the condition of the stools following them. Has lost 50 pounds in the last four years.

COMMENTS AND OPERATION

This patient's trouble has been mostly distress after meals. He has lost 50 pounds in weight. He is a young man. He leads a very irregular life. He is an Assyrian. He travels all over the country, and eats anywhere and everywhere, wherever he happens to be, so that his method of living has been irregular.

We go through the rectus sheath on right side, close to the midline, and it is receding toward the central side. We separate the muscle entirely from the median line and displace its fibers outward, so that the plain of division of the muscle and the plain of division of the aponeurosis are on different lines, and in that way we lessen the likelihood of having hernia or stretching. You see, there is the linea transversus recti. As we never cut between the forceps, we are not so likely to cut the intestine or the omentum. Cutting it on the sloping side of the forceps elevation the air rushes in at once. Here is the stomach in the field, and there is the gastroduodenal junction. There is the large duodenum. The pylorus is patent. There are no adhesions on the surface of the pylorus. Here is the gall-bladder.

Let the record show that the gall-bladder is easily compressible; that there are no adhesions of the gall-bladder; that there are no stones in the gall-bladder, and that the color of the gall-bladder is not altered; it does become altered when you have an infection. This is not an infected gall-bladder—you put that out of consideration. There are no stones in the gall-bladder.

Let us commence and go over to the left on the stomach. We have no evidence of blood in the stools other than the statement that he had a dark-colored stool, which we do not attribute to blood, but to medication. There is no palpable ulcer in the ulcer zone of the stomach clear up to the cardia. I now have the pylorus in my hand. You see how nicely that zone can be exposed. You can lift it clear up into the field. I find that the duodenum is enlarged, but there is no cicatricial tissue that I can recognize as evidence of a previous ulcer of the duodenum. There is no stellate scar, a finding first accentuated by Mayo and Moynihan, on its surface. There is a firm band over the duodenum which may be likened to a violin string; it crosses the duodenum below and compresses it; this, I think, accounts for the symptoms. It is as strong as a violin string and goes right across the duodenum. Whether that is producing dilatation of the duodenum we do not know. It is evidently a connective-tissue band. It goes clear back to the liver, and is attached near the neck of the gall-bladder onto the liver. We will sever it and free it entirely. It is unquestionably a connective-tissue band, but it is not enough to satisfy one that it is the cause of the dilatation of the duodenum. The duodenum is exposed, and all the remaining portion of it comes right up into the field. There is the dilated portion above; there is the commissure over the head of the pancreas, where it was compressed and where it was held down.

In analyzing megaduodenum you have a picture on the wall showing what occurred in Rokitansky's condition, who described it in his work on pathology, and who believed that it was due to some cicatricial formation. A subsequent theory of its production was that it is due to the superior mesenteric artery compressing the first portion of the duodenum. In our analysis of the cases that we have had the latter has not been so at all. It has not been the superior mesenteric artery: it is an angulation. As the duodenum ascends in these cases (and not in the normal anatomic relation), to pass under the great mesentery, it is bound down by a band or short fold of firm peritoneum just to the right of the superior mesenteric artery. This obstructs it, and when the mesenteric artery is elevated, the duodenum is not relieved in the least. It comes up as a firm band holding it, and when the duodenum distends, it rolls around forward on that band. You see how little effort there is required to produce great obstruction. We have had a number of these cases, and in each one we have been able to demonstrate this fixation. In each one we were able to demonstrate that it was not the superior mesenteric artery that caused the compression, but a peritoneal fold or band.

Let us see how the jejunum is below. Let us see whether we have any other fixations here to cause the trouble. The next thing to do is to turn the large intestine forward and get the first portion of the jejunum, which is always over here in the neighborhood of the pole of the left kidney. We have no trouble at all in bringing it out. There is the ligament of Treitz. The jejunum comes out in the regular way. That brings to mind the fact that we have had two cases of ulceration here below the mesocolon in the first portion of the jejunum, just below where the jejunum has been crossed by the epigastric artery. In one of these cases we have had a lateral anastomosis from ulceration and transverse pathologic perforation. We will go down and see whether we have any trouble in the right lower quadrant of the abdomen to account for this condition. We hook up the large intestine; we bring up the caput coli into the field and endeavor to locate the meso-ileum. There is the ileocecal valve. Here is the appendix. The meso-ileum is fairly long. There is the ileum coming up, and here is the appendix shining through. It does not come up. Let us see why it does not come up. Like an eel, it slips away. It is embedded; it is hooked around the meso-ileum and passes over the iliac vessel. Let me see if I can get the end freed and bring it up without enlarging the incision downward. There is the base of it, but I find I cannot bring it up. I am freeing the adhesions around in the neighborhood cautiously. I think I can free it and bring it out. Here is the caput coli up again. It starts across in the direction of the midline; in place of coming down toward the fossa it comes over to the median line. I am ready now to swing it out. It is in a pathologic condition. Look at it! It is in a chronic distended, greatly infiltrated condition. This brings up the point I have previously made, namely, the difficulty in making a differential diagnosis even when you have a patient that you can talk to and from whom you can get the history. Here is the pathologic appendix, and it has pathologic adhesion.

Let the record show that the only lesion discernible in the upper portion of the abdomen was a band compressing the duodenum; that above that band there was pronounced dilatation; that this band extended back and became adherent to the right lobe of the liver just below the gall-bladder. Let the record further show that there was no evidence of a duodenal ulcer, as we had rather concluded that it was more likely to be a duodenal ulcer than a stomach ulcer, because the distress did not come on until an hour and a half or two hours after his meals.

I am ligating the mesenteric artery in the appendix and overlapping the peritoneum. In putting in an overstitch suture in that way it ligates the mesenteric artery, and, at the same time, makes a complete circle of peritoneum around the appendix. The next thing to do is to tie the appendix. This ligature on the appendix I put into a crease where I compress the appendix with a hemostat. Now I will grasp the appendix with forceps and cut it off. This is the ligature on the stump. We are ready for the inversion of the stump, and as we invert it we pull the puckering string taut. That puckering string is essentially a Pagenstecher linen Lembert suture. In addition to this we put in another row of Lembert sutures, so that we have three sources of protection, two of which are probably unnecessary, but always secure and practically the same as one would close a bullet perforation in the same locality in the bowel.

Formerly we just tied the appendix with catgut, and later we amputated it, using one single row of catgut sutures. It occurred to one of the best operators in the world, who did the operation in that way, to put his catgut stitch through the mucosa, so the catgut came in contact with the intestinal juices. It was digested, and several hours after the operation the patient had evidences of perforative peritonitis. He was a good surgeon. Notwithstanding it was a simple interval operation he recognized that something had gone wrong. He immediately opened the abdomen and found the catgut had been digested, and there was a leakage of the intestinal contents into the peritoneal cavity. He closed the opening again with Pagenstecher linen and the patient recovered. Before that time we always used catgut in our closure, namely, we put in that puckering string of catgut. From that time we have used Pagenstecher linen. If such an accident occurred in the practice of that surgeon, a similar one could occur to any surgeon. In using Pagenstecher linen you have none of the chances of trouble: if it does go through the mucosa, it has no capillarity, it does not carry infection along its lines. In an early article which I wrote in connection with intestinal suture I accentuated the fact that if the needle went through the mucosa, it was fatal, but since we have been using Pagenstecher linen in a very large percentage of cases of gastro-enterostomy, we have not had this fear. If you put the needle in with tension on some portion of the intestine, it cuts a hole before the peritoneum agglutinates and before it has time to cover up there may be leakage and infection.

This patient had never had a hemorrhage. His distress came on from one to two hours after meals, when the food began to go out of the stomach. This is true of lesions that occur, first, in the pyloric zone of the stomach; second, of ulcers of the duodenum, and, third, of lesions caused by irritation farther down in the tract and in the appendix. It is at the time the material escapes from the stomach that it produces trouble.

In the pathology let the record further show that there had been a previous appendicitis, although we were not able to elicit any history of an acute inflammatory attack; that there was fixation of the appendix across the iliac vessel, not in the great mesentery.

That is one of the positions in which, when you have an abscess around the appendix, the abscess appears in the pelvis or at the lower pole of the left kidney. It is, fortunately, a rare condition. It is one of the dangerous elements, however, where you have peritonitis following appendicitis, because you have pus extending along up behind the mesentery, and the only wonder is that it does not occur more frequently when you think of the number of times the appendix hangs over the iliac vessel in the pelvis and therefore is under the great mesentery. As the mesentery is attached it passes from the right iliac synchondrosis up to the left side of the second lumbar vertebra. The mesentery is attached above, and still, when you look over the literature, you see how few cases we have had of pus appearing at the pole of the left kidney. In children we more frequently have pus appearing in the pelvis and the secondary abscess follows along the sigmoid into the left side. We have it appearing in 5 per cent. of the cases when we operate in the pelvic abscess zone. In other words, 5 per cent. of the cases had abscess in the left side, and the abscess in the left side bore the same relation in position to the left anterior superior iliac spine that the appendix abscess did to the right. There is a definite anatomic reason for this.

When abscesses form from a rupture of the appendix in the pelvis, the pus takes one of two positions: It goes onto the inner central side of the sigmoid, or if it has been deeper, it passes out and goes on the outer side and appears near the anterior superior iliac spine. There was a time when the indications were to cut in and open the abscesses, and if you will look over the literature, you will find that in a number of instances fecal fistula resulted from the opening of abscesses on the left side. Fecal fistula was attributed to the fact that the bowel was ulcerated by the presence of pus. No, that is not right. The bowel was not ulcerated from the presence of pus. But if it were the central type of abscesses and you opened them from the left iliac fossa inward, you had to go directly through the flattened sigmoid to get to the abscess, while in the peripheral type of abscess you open at once into the pus and do not get into the bowel.

In an article on Appendicitis which we wrote for Keen's "System of Surgery" we pointed out that there was a definite anatomic reason why an abscess appeared in one position and why it appeared in the other, and we had to know when it appeared in a certain position before we opened it. Patients now are operated on early; they are operated on usually before peri-appendicular abscess forms, and the dictum we laid down at the International Medical Congress held at Budapest, in discussing this subject, was this, that "every case of acute appendicitis that comes to the doctor with the abscess outside of the appendix has in it unquestionable proof of the inefficiency of treatment up to that time." No explanation, nothing, fixes it up. When the abscess is outside of the appendix, it is proof that the physician waited beyond the regular limited time, and it is proof that he hazarded the patient. There was a time in the early history of appendicitis operations, if we could let out a quart of pus we became exultant over it. *We thought it was a great find; but we hide that now.* We are no longer proud of such a find. We do not see large quantities of pus any more in cases of appendicitis. It is not a proof of our efficiency any more, and we are, fortunately, rapidly coming to the time when we do not have these large products as a sequence of unpardonable delay—miscalled conservatism—truly ignorance or egotism.

The only places yet in the acute infection where we are behind in the time limit of treatment are in the joint infections and bone infections. We are too far behind in these two. Joint infection must be relieved early, or the synovial membrane is going to be destroyed. We must let the pressure off. In bone the maximum destruction or maximum necrosis is reached inside of forty-eight hours; therefore if we are going to be of great value, we must get these cases inside of the first twenty-four hours. We should be called upon as early in cases of osteomyelitis as we are in cases of appendicitis, because these patients have great pain, they have delirium, they are intensely sick, and they should be operated at as early a date as possible. To let the disease go on and then remove a large fragment of bone is nothing to be proud of; it is just the opposite: it is a stigma of incompetence.

PLASTIC OPERATION ON THE FACE

COMMENTS AND OPERATION

This patient was operated on originally for carcinoma of the lip seven years ago. A second operation was performed in 1907 or 1908. The patient has had three operations, one of which was for epithelioma of the lip. At one operation we took out the glands on both sides of the neck. The disease returned in the skin. We followed that line, and, as you see, the flaps were twisted over from the neck onto his face. He has had no evidence of a return *in loco* for several years. That is a fairly good result.

We have heard a good deal recently and for a considerable time about the cases of cancer that have been diagnosed early and operated upon promptly, and the number of cures that have been brought about. Now this is all very nice, but "early" does not mean much in cancer when you take the statistics of one of the large London hospitals, collected two and a half or three years ago, and find that the final cancer mortality of epithelioma of the lip is 52 per cent. Epithelioma of the lip, as you know, is exposed to the patients and to the medical profession from the beginning, and is considered less malignant than cancer elsewhere, and yet it has a cancer mortality of 52 per cent. when removed before the glands are perceptibly affected, and about 76 per cent. when the glands were affected at the primary operation and removed. So high a mortality is shocking, and when we are struggling to take out cancer of the esophagus and cancer of the cardia of the stomach, it always makes me think of that hymn, "There are heathens nearer home." And it is a fact, there are conditions so much nearer home, if we would only concentrate our attention on endeavoring to determine if there is any possible means by which we can estimate what an early period in cancer is.

Whether the cancer be due to a bacillus or to a protozoal cell it does not matter so much, from a surgical standpoint, as it does to know how soon the epithelial protozoal cell, after it leaves the primary focus of penetration of the basement-membrane, goes into the first gland. When that is determined, then you can positively and honestly say this cancer is late or that one is early. When you get a streptococcic infection it goes quickly through the circulation or lymph-spaces or it never goes. The staphylococcus, on the other hand, can be present for days and days before going into the lymph-spaces and disseminating itself. When these cases of cancer are diagnosed early and operated promptly, we have clinically a greater number of cures. I would not admit this without very grave question. We do know that there is a type of cancer that produces a firm, compact coffer-damming, which means, what? Firm, compact connective-tissue response in the way of hemming in and holding in and surrounding and retaining these erratic epithelial cells; that these remain *in loco* longer than the other variety which grows rapidly and have no connective-tissue coffer-damming and protection: just exactly the same as in the other varieties of infection.

We took out this patient's glands, and we had no manifestation of the disease below that at any time. We had a return of the disease in the skin, but not in the glandular system, which showed that the cancer-cells had gone into the lymphatics in the neighborhood and had remained there and produced cancer there, but did not go beyond the glands we took out. He came back with another lesion, namely, with an epithelioma of the side of his nose, entirely independent, and I brought him in here to show you how we handle this class of cases. We simply made a clean dissection and took all the thickness of the derma there from his arm, freed an equal sized and similar shaped piece of skin, detached it, fixed it in accurately with sutures, and quilted the base of the derma to the connective tissue beneath. If you do not quilt it to the base you will have an accumulation of blood between the flap and the tissue will die. After you have quilted it to the base, you commence to get early healing—nutrition by osmosis. Connective tissue does not heal by osmosis. Connective tissue heals by direct circulatory supply. The skin, cartilage, and cornea are the great osmotic tissues of the body—the skin, the least of the three. In another operation we made a large transplantation—where I took two-thirds of the derma and transplanted it—though it healed in nicely, it contracted. The graft I took previously on the same plan lived and did not contract more than 15 or 20 per cent. This lived, and contracted 40 per cent. We will have to put in a little more next time and give greater looseness to it. In the next case or operation I will take the full thickness of the derma in place of two-thirds. The advantage of an attached transplant is that it fills in the space and subsequently stretches or enlarges; it has greater sensation than normal skin—that is one of the peculiarities of it.

CYST IN THE LEFT ILIAC FOSSA

HISTORY

Female; age, forty-seven; housewife. Has been married for twenty-eight years. Born in Ireland. Uses tea and coffee in moderation. Drinks an occasional glass of whisky. Patient was admitted to the hospital on account of a swelling in the left lower pelvic region, just internal to the spine of the ilium. Family history negative. Had the ordinary diseases of childhood, but has had nothing since until the onset of the present trouble. Has borne eight normal children, all normal deliveries. No miscarriages. Venereal history negative. Her menstruation had been of the twenty-eight-day type, three- to five-day flow, four napkins a day, previous to the onset of her present trouble.

Present Trouble.—Commenced in August, 1907. The patient states that at this time she noticed an increase in her flow. She flowed from ten days to two weeks, and at each time this flow continued to increase in duration and intensity for the following two years, and was accompanied by dragging pains through the pelvis and indigestion. Her heart would palpitate frequently and she became anemic and thin. In June, 1909, a hysterectomy was performed and a large fibroid of the submucous type removed. The incision drained pus after four days following the operation up to fourteen days. The wound was completely healed in three weeks, and never caused any more trouble. On September 20, 1911, the patient states that the abdomen just internal to the hip-joint became swollen. The swelling was circumscribed, protruded like the half of a sphere 2½ inches, and was tender and painful on movement. The growth became no larger and gave no more trouble after October 10th until December 3d, when it began to pain again and grew very rapidly, reaching its present size in one week. During the last three weeks it has remained the same size, but is still painful at intervals. The patient states that aside from the discomfort caused by the swelling she has felt very well.

COMMENTS AND OPERATION

This case is interesting, the patient being admitted on account of a swelling in the pelvic region. We do not know whether the ovaries were removed at the time she had a hysterectomy performed or not. That is proposition No. 1. She had a suppuration which lasted only a short time and disappeared entirely. She finally, in September, 1911, began to have a tumor in the left iliac fossa the size of an egg or a billiard-ball, or half of that size, and then from that time it has suddenly increased in size. It became painful and has remained at that increased size up to the present time. It fluctuates. It is fixed to the fossa, and the question arises, what is it? It was aspirated and grumous material was drawn from it, and, according to microscopic examination, it is the result of a hemorrhage, and, in addition to this, it is said, there were epithelial cells of the large type in the fluid. If this is so, it may be a grumous cyst from the ovary. The question is, when the hysterectomy was performed, what was done? Did they let the ovaries recede to the margin of the pelvic wall and pack them off, so that the cyst was fixed to the pelvic wall, and has it developed there? If it is not a cyst of the ovary, what is it? Is it a carcinoma, or is it a sarcoma in which a hemorrhage has taken place?

From our interpretation it looks like a cyst that has developed in the ovary following operation, with the one exception that it is too far to the side of the pelvis, and if it had not been for the fact that she had a laparotomy and the uterus was removed, we would say at once that it was of this origin. What we fear is that, with a tumor in this position, it may be a sarcoma. One of the things I thought it might be was an occluded ureter. If she had occlusion of the ureter, this could be a development from the occluded ureter, but the aspiration settled this question negatively. It is grumous material that came from it, and the enlargement was sudden. You see that comes away out and looks as though it originated in the ileum. It is fixed at its base. We do not feel the iliac vessels in front of it. We could not palpate them in front of it, so we do not know just where they are. Both rectal and vaginal examinations are negative. It is a part of the bone, so far as we are able to make out. It is immovable, but it may have originated in the ovary. If it is a sarcoma, we will close it up and run away. It extends pretty well above the crest of the ilium, and still it does not go up into the renal fossa to include the kidney in the enlargement.

We are going through the linea semilunaris, and there it is. There, you see, is the outer margin of the rectus muscle; you see the aponeurosis of the external oblique, and here is the inner layer of the rectus showing. We are opening it in that line to be perfectly free for subsequent work. Here is the peritoneum under the rectus muscle. There are the two sheaths of the rectus held separately. We are just at the margin of the peritoneal attachment to the tumor. It is a secondary neoplasm, I think. That it is a metastatic neoplasm you see at once. No examination microscopically was made of this fibroma, or whatever it was, that was removed.

Here is a neoplasm very similar to a cyst in the pelvis. It is a sarcoma or a carcinoma. There are metastatic deposits in the omentum. That makes me believe it is not a sarcoma, but a carcinoma, because metastatic carcinomata are occasionally cystic—the squamous epithelial type much more frequently than the columnar epithelial type gives cystic metastases, as we see them in the axilla, neck, etc. There is the tube. Here is the fimbriated end of the tube in good position, and here is the ovary, and this appears to have originated in the ovary.

Here is another one running across from the other side of the same type and consistence. The glands run up behind the small intestine and are somewhat enlarged. I find a regular rosary of them running in an upward direction. Here is another one away up behind, situated retroperitoneally, on a level with the pancreas. The liver is free. It is non-adherent. There are no nodules in it. We have metastatic malignant neoplasms all through the lymph-glands, probably having originated from an original growth that developed into the broad ligament, and from that spread through into both sides of the pelvis and abdomen subperitoneally through the lymphatic spaces.

I started to speak about the cysts. We are told that this cyst had epithelial cells in it. We begin to think that the pathologist is probably right in saying that he found epithelial cells. We not infrequently find in the epitrochlear gland cystic carcinoma; we not infrequently have cystic carcinoma in the axillary space. Again, we have carcinomata that occur on the feet; we not infrequently have cystic carcinomata in the groin, the lymphatic glands, with a large quantity of fluid—sometimes a cupful of it—in one of these cysts. You aspirate, and you find epithelial cells in the metastatic process. Just as soon as you dissect it out you find it has a thick wall and is full of fluid in the center. This retroperitoneal cyst, which involves one of the retroperitoneal glands, I interpret to be of carcinomatous type that is undergoing cystic enlargement. There was a hemorrhage into it at one time. The glands I feel are all retroperitoneal. This cyst is in the direct line of lymphatic transmission, as are all the others I have mentioned. This brings out a point that was made by Bland-Sutton in his examination of uteri removed for carcinoma. He [Bland-Sutton], in his London experience, in a large number of cases of uteri removed at the London hospitals, showed that 4 per cent. of all the cases of fibroma had carcinomata present in the mucous membrane of the uterus, as well as a fibroma in the connective-tissue structures of the uterus. We have always estimated that this was a very large percentage for this country, and the explanation which we give was that in London fibromata are not removed as early as they are in this country. Here they are removed as soon as the diagnosis is made. There they are not removed until they attain great size and the patients become advanced in years, are therefore in the carcinoma zone of their evolution, and they are more likely to have them. In our experience in this country we have only had a few cases; it would not represent 1 per cent. of our cases in which we have discovered carcinoma associated with fibroma.

It is true we may have overlooked carcinomata because there is no doubt that we did not make as careful examination of all uteri we have removed as was made by Bland-Sutton when he was carrying on this investigation. But it emphasizes the fact that the uterus with a fibroma can have associated with it carcinoma of the mucosa, and this element is to be kept in mind when a hysterectomy is to be performed. Knowing, as we do, that fibroma of the uterus occurs in the fundus and that a fundus carcinoma is very rare, that is another reason for believing that it is not so common as he interprets it. A fundus carcinoma is one of the very rare lesions. I could count all the fundus carcinomata I have seen in this large period of time on the fingers of one hand. So you see it is a very small number for such a long period of time. In those cases we have had, the conditions have been exceptionally favorable for operation. The results have been good. In one of my cases it will be eight years this coming June since I operated, and that patient is living today. In that case the tumor was so rotten that when I put on the vulsellum the whole fundus came out. I dug out the base. I have not one patient on whom I did a hysterectomy for carcinoma of the cervix who is alive two years after the operation without a return. The fact is that in the fundus of the uterus you have practically a low lymphatic circulation and late or no metastasis. This is true of the fundus of every organ—the fundus of the stomach, the fundus of the gall-bladder, and the fundus of the urinary bladder. It is also true that the fundus of every organ is rarely attacked with carcinoma, and when it is attacked, you have the best conditions for permanent cure by excision from the fact that you have no lymphatic circulation. That has been our explanation of it. When you come to the cervical zone of every organ, like the cervix of the uterus, the cervix of the urinary bladder, and that of the stomach, which is the pyloric zone,—these all have a rich lymphatic supply,—there is early transmission through metastasis, which usually leads to fatal termination. My experience with not having a case of columnar-celled carcinoma of the uterus living two years is not unique. When Professor Pozzi, of Paris, was here on his last visit, I asked him and Dr. Byford of this city, at a luncheon, how many cases of carcinoma of the cervix each had had that had been *referred* to them for operation. They could count them on the fingers of either hand. That gives you an idea of how rarely the columnar type of carcinoma is cured, even with a hysterectomy and in very good hands.

From the statistics we have very good reason to hope that the new operation of Wertheim, which consists in taking out all the glands and taking out all the uterus and cleaning everything up to the kidney level, is going to give better results. It is based on the statistics up to date, but the statistics of every operation for carcinoma always meet the same fate: fine in the beginning, medium in the middle, with a quick return in the end, to the average small percentage of permanent cures; and that applies to Halsted's breast operation, just as well as to others. And this is not surprising either when we are told that in the least malignant of all the carcinomatous neoplasms, namely, epithelioma of the lip, according to the reports of the London hospitals, 52 per cent. of all those that were operated upon before the glands became especially involved in the neck would die of carcinoma; but ultimately 76 per cent. of them who had enlargement of the glands at the time of the primary operation in which the glands were removed died a carcinomatous death. Here is a lesion open to the observation of the profession from its earliest stage, considered the least malignant of all, and yet look at the frightful cancer mortality! It shows what a hopeless proposition we have to contend with and how discouraging it is.

The Wertheim operation will meet the same fate that all the extensive operations have met as soon as time elapses. Why do I make this statement when I have had no experience with the Wertheim operation? You may say that I am not justified in making such a statement when the procedure involves other than the well-known route of operation. The typical, classic, pathologic route of the cancer lesions, with its malignancy and their tendencies to recurrence, are not changed by the route or extent of the operation. Why? The nature of a dog is not changed by the muzzle. He is just the same willing and vicious animal, if he is vicious, no matter what muzzle you put on him.

We know that: we are unable to muzzle the malignant epithelial cells. We know further that we have no justifiable data for assuming that we know the period of time that elapses from the moment the first erratic epithelial cell penetrates the basement-membrane until it is transmitted to the next lymphatic gland, nor to the second lymphatic gland, nor to the third lymphatic gland, nor to the thoracic duct, nor to the general circulation from that point, because we all know, particularly those who have had experience, that in amputations of the breast as well as in amputations of the uterus,—and particularly of the breast,—within a few weeks after operation, with no apparent glandular enlargement, and in many instances at the time of your operation, or shortly after the recognition of the lesion, you have the patient complaining of pains in the osseous system due to metastases. I mention that particularly because that is where metastases take place from the breast most frequently, excluding the glands. You have these present at the *time* of the operation and the metastases commencing in tissue *before* you operate. Therefore, chasing after these few lymphatic glands and removing them has not materially altered the situation, as has been shown particularly by Halsted's statistics in operations upon the breast. If you read Halsted's paper, which was read at the Cleveland meeting of the American Surgical Association, you will find that when you exclude the cases in which there was no demonstrable enlargement or demonstrable metastasis, as determined by microscopic examination at the time of the primary operation, you have a mighty small percentage of permanent cures, even with the extensive operations. We warmly advocate the glandular removals, but deplore the final results.

TRAUMA OF CYSTADENOMA OF THE BREAST

HISTORY

Female; patient is a governess, aged twenty-six, who was admitted because of a small nodular mass in the left breast. Family history negative—no tuberculosis or malignancy. Had the ordinary diseases of childhood. Has been well since. Venereal negative. Menstruation slightly irregular—every four or five weeks—five days, four napkins a day. Dysmenorrhea on first day.

Present Trouble.—Patient states that four weeks ago she was hurt on the left breast by being kicked by a baby. This was on December 7th, and December 27th, twenty days later, while washing, she noticed a slight nodular enlargement on the left breast. This is the size of a large marble. The skin is movable over the nodule, and the nodular mass is freely movable in the breast tissue. There is no infiltration in the surrounding structures. There is no glandular enlargement in the axilla. There is no hyperemia at the site of the nodule, and it has never been tender on pressure or given pain.

COMMENTS AND OPERATION

This patient was playing with a baby and the baby kicked her in the breast; shortly after this she noticed a nodule in the breast about the size of a large marble. She took very little notice of this at the time, notwithstanding the fact that she felt soreness in the breast for a number of days, and from her statement it can be adduced that it was not a primary hematoma of the breast. Therefore, if it was not a primary hematoma of the breast, the question arises, what is it now? The next question you ask is, before answering that, whether there was really a tumor present at the time she was kicked, and was this kick a coincidence that has disturbed the tumor already existing? If there was no tumor present at the time she was kicked, then what is it? It is either a traumatic cyst or it is a sarcoma, because carcinoma does not come in so short a period of time following a trauma.

The breast is the one place in which a *single* trauma of moderate severity produces a carcinoma, and, besides, is the only place in which we have carcinoma due also to chronic irritation as well. We have carcinoma occurring as the result of, or associated with, mild trauma or repeated irritations. But in the breast one or two or twenty years after there may develop a malignant lesion at the point of trauma.

My interpretation of this case is that it is a cyst, and that it was present before the baby kicked the patient. In the first place, the mass is too movable for it to be a sarcoma. It is too movable for it to be an inflammatory infiltrate. It is too movable for it to be a hemorrhagic infiltrate. It might possibly be an encapsulated sarcoma or an encapsulated adenoma that was present before she received the kick. It may be an encapsulated cystadenoma, but then it preceded the injury. But the peculiar part is that if it was there before she received the injury, she did not notice it at the time of the injury.

I have now opened this cyst, and you can see the exudate coming out from it—it is an encapsulated tumor. It looks as if the trauma she received started this on a malignant career. There, you see, is the papilloma starting to grow in it; it looks, as I have said, like a papillomatous cyst of the breast starting on a bad career. It does not look like a sarcoma. It is four weeks since the accident. This tumor or cyst has not developed since that time, because this fibrous capsule condition could not have developed in that length of time. It is a papillomatous cyst, with possibly a beginning malignancy at the base of the papilloma—an epithelial malignancy.

Let the record show that our interpretation is that she had a cystadenoma at the time of the accident; that the accident has started up a rapid secretion in the cyst and possibly started the papilloma; that when she noticed it at first it was as large as a hazelnut, but now it is as large as four hazelnuts in its capsule; that it was encapsulated; that all the capsule and a portion of the breast adjoining it were removed; that it was on the anterior portion of the mammary gland and near the nipple margin.

[The microscopist's report on this case was fibro-adenoma.]

ANASTOMOSIS OF THE EXTERNAL POPLITEAL NERVE

HISTORY

Male; age, thirty-five; married. Admitted to hospital September 15th. Section hand by occupation. Has been in the United States about five months. Had the ordinary childhood diseases, with good recovery. Ten years ago had malaria lasting four months, chills every other day, fever, and drenching sweats. No other adult disease. No abscessed tonsils; no bad teeth. No specific history or tubercular history.

Present Trouble.—Two months ago patient had pain in the left knee, coming on gradually, and not very severe, but in six days it became so severe he had to quit work and go to bed. On the sixth day he had a chill lasting one hour, and repeated several times during the day for shorter periods, and had temperature. Has had chills and fever since leg or knee began to swell, and there was no redness, but it was very tender to touch. Could not endure bed-clothes on it or bear any weight on it. At the time of temperature and chills leg began to get stiff, and he held leg quiet on account of pain. September 11th he was operated on, but does not know the nature of the operation; no history of injury.

COMMENTS AND OPERATION

Dr. Murphy: Did the patient have a chill or pain first?

The Interne: Pain first.

Dr. Murphy: Then the history is not correct. This is an example of the way most histories are taken in hospitals. When a history is inaccurate, its value is destroyed. A good history is more valuable in making a diagnosis than all other things combined. The physical findings and clinical course, outside of the history, are merely confirmatory, while the history is the pivotal point on which all diagnoses should be based, and the man who wants to make good diagnoses as an internist should read the old edition of Trousseau and review the clinical histories of diseases which he wrote in his time. Trousseau knew little or nothing about the pathology of disease, but he was such a keen, close, accurate observer of clinical phenomena in the order in which they occurred, as associated with the physical appearance, the secretions, and so on, that men of intelligence could make pathologic diagnoses half a century after the book was written. And this is a point we wish to accentuate.

According to the history that was read here, this patient had pain and then a chill. That is not the way the trouble begins. The chill comes first, because it is a manifestation of metastatic infection. The chill comes when the septic material is in the circulation, and the precipitation and subsequent increase of the septic material give the pain. He may have had a second or a third chill, depending upon the amount of reproduction and absorption of the microbic products that took place at the focus of precipitation.

This man had an infection in his knee. The doctor who first saw him said the knee was inflamed; it was swollen badly, was painful, and he opened into the knee and inserted a drain, and the patient came to us with his knee opened. It had been opened the day previously. We immediately exposed the freshened wound, united the capsule of the joint and skin with sutures, and filled the knee with 25 c.c. of a 2 per cent. solution of formalin and glycerin, as it was suppurating. We put on an extension; we aspirated and injected again the same amount ten days afterward. I want to show you that the knee is perfectly good; that it has not been hurt at all, notwithstanding the fact that it was opened and drained for twenty-four hours. Look at the perfect motion he has! We filled the knee twice with injections of formalin and glycerin. This was done two months ago. There was a second incision made in the previous operation over the head of the fibula, evidently expecting to find pus outside of the joint, but instead they cut the external popliteal nerve.

I am glad to have such a case to present to you, because it shows what can occur with infection and the joint opened when it is properly handled.

The external popliteal nerve is the motor nerve of all the extensors of the foot. At the previous operation they cut it off in a place where it is going to be difficult for me to find the distal portion. I am going to try to find it, however. The proximal portion I should have less trouble in finding. It goes along behind the head of the fibula. You can feel it slip below the head of the fibula at your own knee. I can feel it in this case, and I always feel it in just the same place. It has already slipped up, and that is why I am going to have trouble in finding enough of any of the distal portion to make an approximation or to get the new nerve axons to come down and form a connection.

This is the third patient we have in the hospital at this time, not counting the one that left yesterday, with nerve lesions. One was a man who had sustained an injury to the anterior portion of his elbow-joint from a steel plate falling across and striking his arm while it was under tension and cutting off all three main motor nerve-trunks, with complete paralysis of the muscles of the forearm and hand. He came in two and a half years after the accident and we were able to reunite the nerves.

I want to see if I can feel anything like a neuroma on the distal end. I rather think I feel it here, and I am going to try to cut into the place where I feel it. As it comes across here [indicating] and becomes the anterior tibial, supplying the extensor group, it spreads at once like the pes anserinus of the facial nerve. In the case above referred to we had to find all three trunks in the cicatricial tissue where the tissues suppurated following the original injury. We made approximation in his median and in his ulnar nerves by elongating with a flap. The musculospiral ends had separated, so that in flexing the forearm we could get contacting without the interposition of a nerve-flap, so that we hoped in this way to get a result, first, from the reëstablishment of the axons, and, second, from the secondary regeneration of the flap that was built over, and in both by a regeneration in the distal axons. It will take fifteen to eighteen months to have motion restored.

There is a mass of fibrous tissue here in the long extensors of the peroneal group that resembles the nerve. It is not the nerve, and it is in the hope that, by keeping close to the bone and avoiding it, I can find the other end, and I am putting in this drop of methylene-blue. It is only a drop, just enough to discolor the muscles in the anatomic position, where it may possibly be found. This proves that my anatomic relations are disturbed because there has been suppuration here. I am using the faradic current to see if any of the fibers of this nerve are intact, and if any of them have recrossed the line of division in the scar tissue resulting from the incision. If any of these fibers are living, we will have a positive response by irritating the proximal side with the faradic current. Bear in mind, in connection with the use of electricity, that, from a surgical standpoint, a living nerve is affected by the faradic current: a dead nerve not at all; that the muscle is caused to contract by the galvanic current, whether the nerve is living or not. It is the simplest proposition in nerve surgery; it is simply a question of physics, if you keep the two elements in mind.

Here is the peroneal nerve. Some of the fibers are regenerated. The peroneal group comes in contact; the anterior tibial portion does not. Here are some of the fibers where they pass off above the line of division. I find the nerve is living. Why? Because the faradic current never produces contraction in the muscles of a nerve that is dead, differing from the galvanic current. Let us see if we can find the distal portion after we go through the cicatricial tissue, and see how much of the distal portion of the nerve is left, and whether we are commencing to get regeneration by means of approximation. This operation requires delicate and careful dissection, and I am afraid you will become impatient, but I will be busy all the time. I find the tibialis anticus does not come into action at all. Its fibers are dead. I am carefully coming over to the distal portion. You see these nerve-fibers. There is the end. There is where they come across to meet the distal portion. A few of the fibers here are regenerated. I must make a splint from here [indicating], crossing this over. I want to show you now the neuroma. See how beautifully it is shown. This nerve is now cut, and we are ready to implant it into the trunk on the proximal side of the point of division. You can see the nerve fasciculi coming right down. One of the nice things about the nerves is that nature has given us many more axons than we really need, and that is why we can cut a nerve and bring the distal portion where we desire it and implant it with perfect safety. It does not matter whether I cut the nerve or not for the immediate effect of faradic response, the distal portion will transmit just the same as it did before, and it will continue while there is a real live nerve to the muscle. It requires some hours for the axons to die after division. Here [indicating] is where I am going to make my line of suture.

We have had some interesting cases in this hospital showing the power of regeneration that exists in a nerve. It is very great. It is surprising how a nerve will endeavor to regenerate if it is given anything like an opportunity, and if the nerves are near together, without the interposition of connective tissue, they will repair very readily and very thoroughly.

I will put in one suture in the nerve-sheath on the inner side just so, in order to take the traction off of the nerve-trunk. This suture does not go through the nerve on either side. It is merely tacked on the sheath to take the tension off. One does not wish to have tension on a nerve at the point of reunion, because with tension there is a likelihood of separation of the axons. You are in an unpleasant zone here on account of the rapid subdivisions and spreading of the nerve. You get the same thing in connection with operations on the facial at the pes anserinus, where you have a spreading out and cannot find enough filaments to make a union. We have no tension on the skin. The nerve scar will be in the soft connective tissue.

Let us go back to the consideration of what we are to expect from this work and when we are to expect it. We have this nerve put out of commission by the surgeon under two conditions—one in which he is positively responsible, and the other in which he is positively but inactively responsible, if I may use that expression, and that is in this way: first, by the cutting, which is inexcusable, and, second, you have the nerve put out of commission with enormously greater frequency in fractures of the femur, in fractures of the leg, and in fractures of the neck of the femur, and in diseases involving the hip-joint. By what? Not by cutting, but by putting on a Buck's extension with adhesive strips passing along and around the prominent portion of the upper end of the fibula held in position by a tight bandage. This produces compression of the external popliteal nerve and results in the drop-foot we used to consider was a part of the results of a fracture of the femur, as was also the talipes equinus. This is not from contracture of the tendo Achillis and the muscles attached thereto, but it is due to a suppression of transmission of the axons to the external popliteal nerve from the bandage and from the plaster too accurately and severely applied around the leg just below the knee. Every one of us has had such cases under observation.

We have in the hospital at the present time a hip case in which the bandage was applied by the interne too tightly, so that when I looked at the case on the third day I found the patient had drop-foot. His nerve was not cut: it was compressed. It was merely a suspension of transmission. If you keep such a bandage, tightly applied, on for ten days or two weeks, the patient can have a neuroma develop in the nerve that would result in permanent paralysis, which means suspension or inhibition of transmission in the axons, which is not an uncommon sequence of these cases. The same holds true when a plaster-of-Paris bandage is applied too tightly around the knee; it is so firmly applied that it presses over the nerve with nothing to protect it. You can feel the exposed portion of the nerve, so that the least firm, fixed, inelastic dressing or substance in the neighborhood is going to incapacitate it. What is going to be the result? When you have a pressure of only temporary duration, you have suspension, and you have restoration of that function fully in six or eight weeks after the pressure is relieved. It is identical with the type of drop-wrist we have had after chloroform anesthesia, when the hands are allowed to rest over the edge of the table and the musculospiral nerve is compressed, and this was formerly considered a chloroform paralysis, while, in fact, it was due to pressure on the nerve on the edge of the table during operation. The medical profession was slow in recognizing the danger of pressure on the external popliteal nerve as it comes over or below the head of the fibula.

What happens when the nerve is cut? The moment it is cut it dooms all the axons in the distal portion, no matter how quickly it is sutured, as the distal axons always die. Whenever a nerve is divided, will it regenerate if the axons are again put in contact? Yes, if the axons have a neurilemma. You know, every nerve axon is made up of three elements: an axis-cylinder, a medullary sheath, and a neurilemma. The neurilemma is the reproductive sheath of the nerve. A nerve that has no neurilemma is never capable of reproduction. All nerve axons have a neurilemma except those in the nerves of special sense and in the true spinal cord.

[Dr. Murphy here continued his talk on Nerve Regeneration, but as this is substantially the same as was printed in the cases of nerve anastomosis published in the February number of the Clinics, it is not repeated.]

VOLUME I *NUMBER 3*

THE
SURGICAL CLINICS
OF
JOHN B. MURPHY, M.D.
AT
MERCY HOSPITAL, CHICAGO

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SURGICAL CLINICS
OF
Volume I JOHN B. MURPHY Number 3

EXHIBITION AT CLINIC OF CASES PREVIOUSLY OPERATED UPON

With Comments, Photographs, and Skiagrams

1. This is a case of bone transplantation, and I am going to make the first dressing. (See Fig. 39.) This was a case of non-union (described in the April number, p. 135). This man came from the Canal Zone with a non-union of his tibia, in which we transplanted a large piece of tibia. The exact dimensions are about ½ by ⅝ by ⅜ inch. That is the size we usually endeavor to get. We took a piece of bone for the transplant from the crest of the other tibia. Let the record show that we have primary union on the side where the fragment was removed. Let the record further show that there is a complete primary union of the wound in the other leg; that the stitches are not disturbed. The wound is in perfect condition up to this time, and as so many days have elapsed with a normal pulse and normal temperature, it should stay in perfect condition.

This dressing is carbolated gauze, and it is only in bone and joint work that we use it. As we have so much oozing, the carbolated gauze takes up secretions which follow operation. If these secretions should decompose when ordinary sterile gauze is used and the air comes in from above or below with motion, the patient gets a saprophytic infection of the skin around the wound, and that may last for days and disturb the skin approximation. If it disturbs the skin approximation, then you can have a deeper disturbance later, and you can have a pyocyaneus infection and other varieties occurring deep in, and in all these cases you have more or less hazarded the flap, and if you have necrosis of the flap, you have exactly the same condition of superficial infection.

2. Here (see Fig. 40) is another case in the same line, un-

united fracture of humerus (described in the April number, p. 193), but a little further advanced. This young man came in with a Lane plate. He had splendid apposition, but not the slightest effort at union. We took out the bone-plate and permitted healing to take place. As soon as healing of the soft parts was perfect, we implanted a large fragment of bone from his tibia, which you see. We put it in the medulla, using the rongeur to ream it out. We took a piece from the crest of the tibia, pushed it away down in the lower fragment, and when we had it ready, we took it with forceps and drove it back up into position, so that it spans the line of fracture. He has an interosseous splint, giving great strength and force, so that in handling the arm at this time you can hardly see that he has any evidence at all of having had a broken arm. It is too early for bony union or regeneration across the line. His musculospiral nerve is not involved at all, although the trouble is in the musculospiral zone. It will take some weeks for that to ossify completely. The Haversian canals in the transplant support the vessels that go above and below from the living bone, and they coalesce across the line, and this fragment will be entirely removed or will disappear, and new bone, first, of that size and shape, will be produced, and then, second, new bone which will take on the size that is necessary for support. There is one, you see, in the tibia. The case came in a rather bad condition. There was a fragment placed across. Here it is before operation with perfect apposition, but no effort at union. Here it is again, as shown in this skiagram, with perfect apposition. Here it is with the wire turned in and the splint inserted. There [exhibiting skiagram] it is with union. We can see no trace whatever of the transplant.

3. Here is a photograph of another case of the same class with the bone-plate in position. We could not swing that around without trauma. We filled in with a graft. Here is a picture showing the graft, very close and very completely [indicating], except the line of periosteum, showing that it is a detriment to the reproduction of bone in this way; it prevents the Haversian vessels from passing from the side through into the

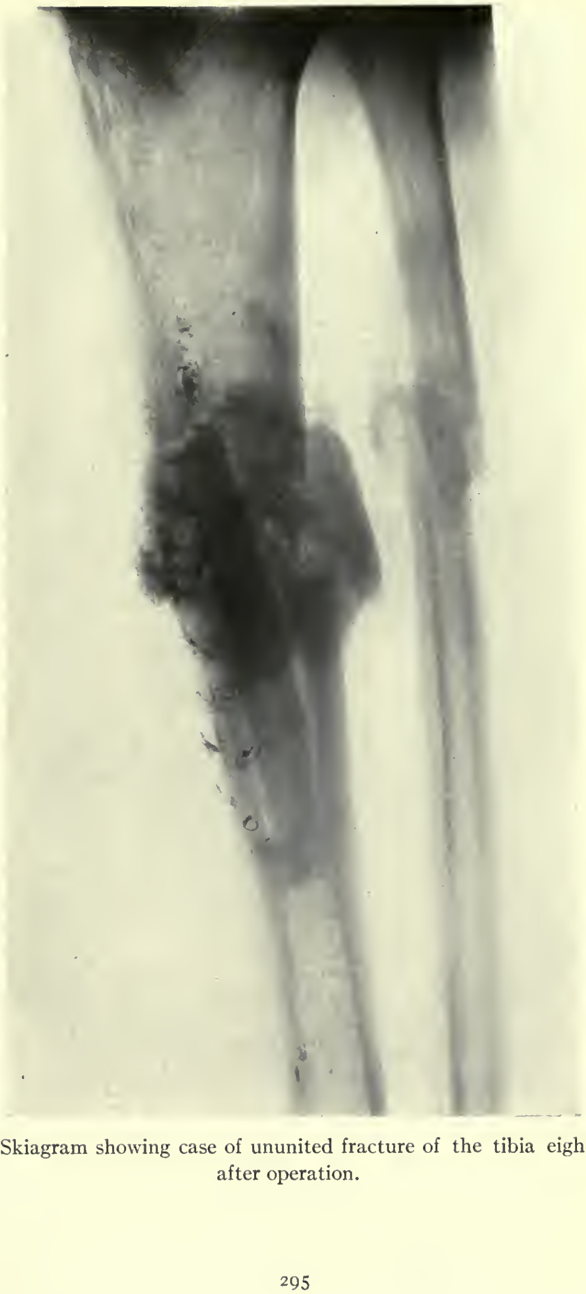


Fig. 39.—Skiagram showing case of ununited fracture of the tibia eighteen days after operation.

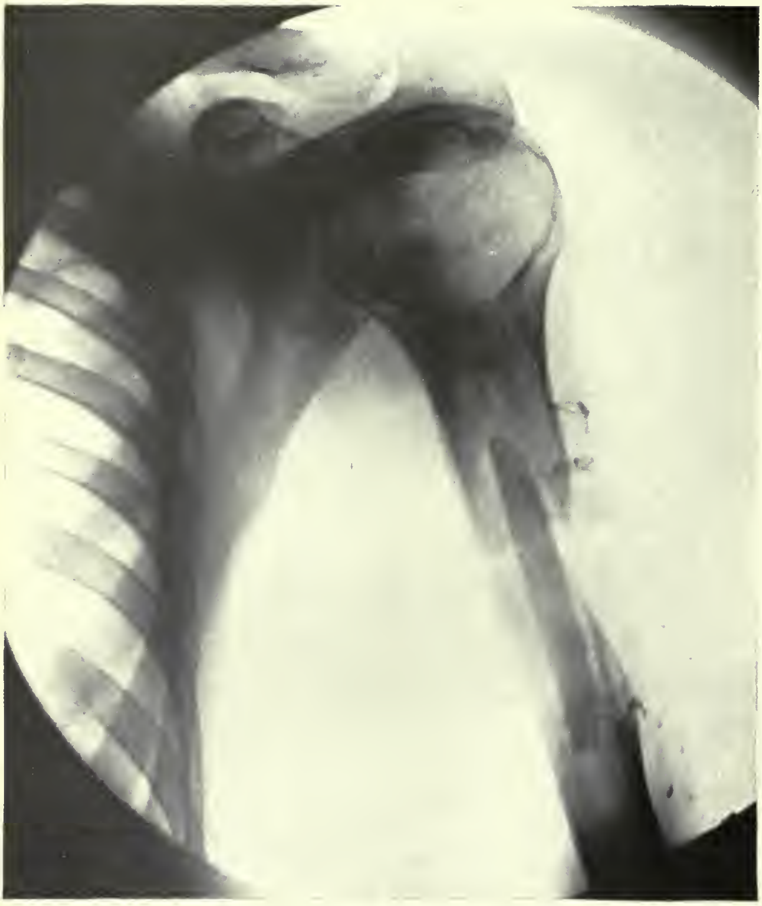


Fig. 40.—Ununited fracture of humerus after operation, showing bone splint transplanted from tibia.

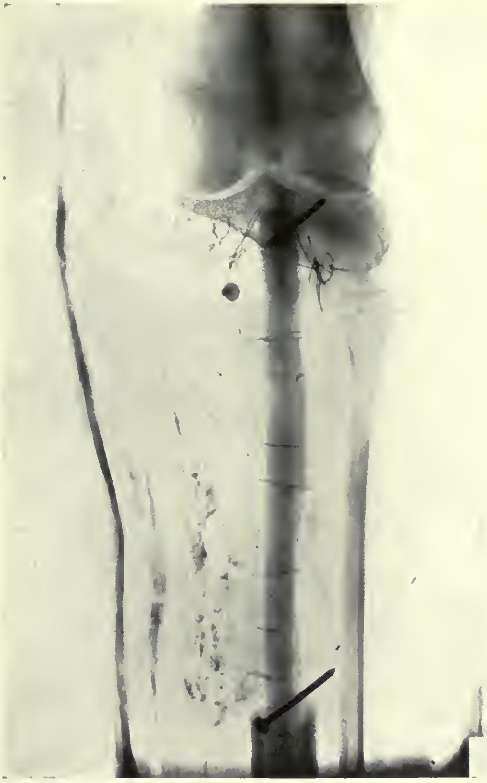


Fig. 41.—Skiagram showing Case No. 4. Sarcoma, involving upper end of tibia. This shows transplanted bone and beginning regeneration.

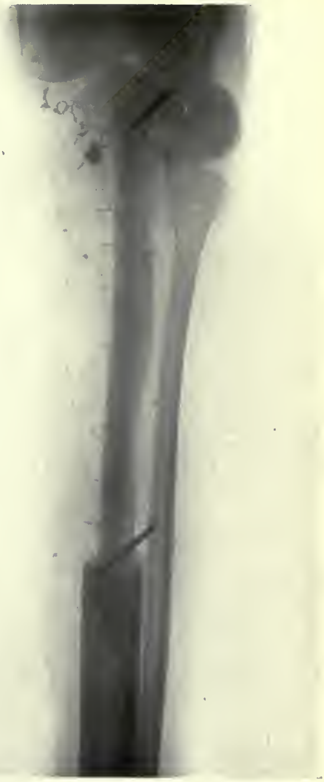


Fig. 42.—Skiagram showing Case No. 4. Sarcoma involving upper end of tibia. This shows a still greater degree of regeneration of transplanted bone.

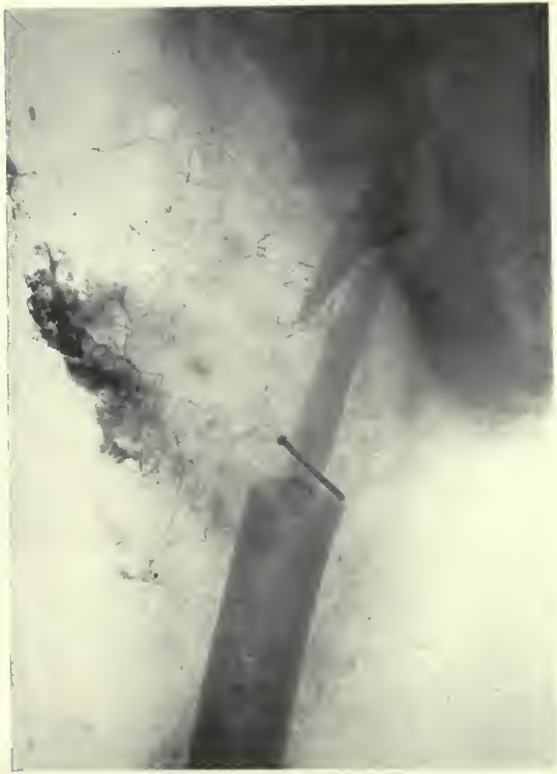


Fig. 43.—Skiagram showing Case No. 5 in exhibition of cases. Sarcoma of the trochanter following an injury. This shows transplantation of bone after resection and its beginning regeneration.

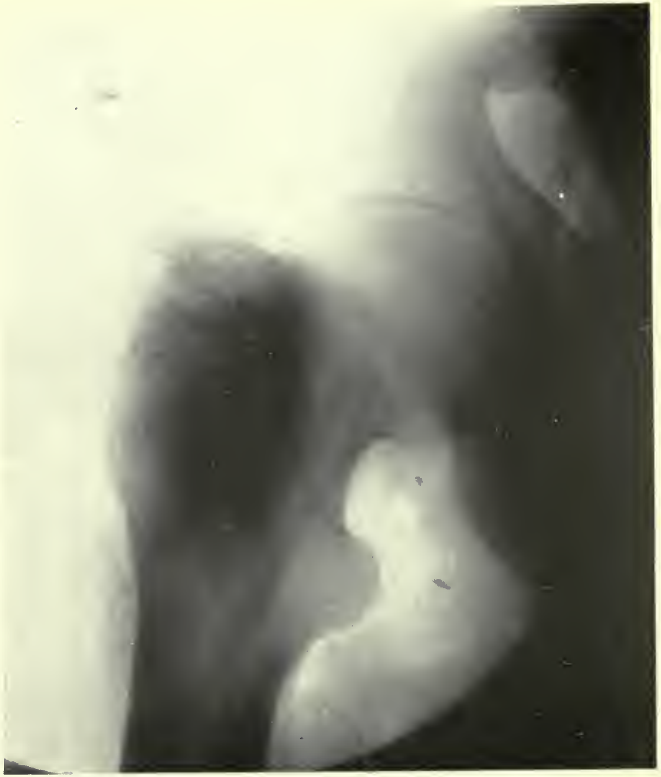


Fig. 44.—Skiagram showing Case No. 5 in exhibit of cases, etc. Sarcoma of the trochanter following an injury. This shows the sarcoma in great trochanter.

Haversian canals, and it looks as if it lived for a much longer time than we believed it would live. We do not know that it reproduces bone here. If you peel the periosteum partially and leave it attached to the shaft of the bone, it will reproduce bone. If you detach it and leave it an inch away, it will produce bone for a time, some of which will remain and some will become absorbed. There is no doubt but that living periosteum has living osteogenetic elements, that it plays a secondary rôle in bone regeneration. In bone transformation you have one requirement, so far as the regeneration of bone is concerned, namely, that the graft contacts accurately with the osteogenetic freshened bony surfaces, either one or two ends. We have here to deal with only one. The rôle it plays is that the vessels pass from the living into the Haversian canal of the dead transplant and regeneration follows. We know that if you put a fragment of bone as large as that into a muscle with the periosteum on, it will all entirely disappear with time. If you will put one end in contact with bone, it will fill up with bone of the size and shape of the graft. It will subsequently somewhat absorb if you have no particular purpose for it in the way of support.

4. This case was one of sarcoma involving the upper end of the tibia. We had just enough of the tibia left beyond the epiphyseal line to be able to cut it off and retain an articular fragment. You see this picture does not show it because it was cartilaginous, and it does not show cartilage at the line of suture. The joint was opened in doing the operation. Bony elements have been reproduced here already. It has smelted into one piece of bone, so that you cannot tell which is the transplant and which is the rest. This operation was done in lieu of an amputation for sarcoma. Formerly, for this type of sarcoma the limb was amputated. This was a slowly growing encapsulated chondrosarcoma, most malignant of this type except the giant-cell. This is the type of sarcoma you have occurring in the mandible, and occasionally in the compact tissue of the tibia and femur, and you have reproduction.

5. Here is another case, the photograph showing involvement of the joint (Figs. 43 and 44). This is a case of sarcoma

of the trochanter following an injury. We took out the upper end, head and all, and transplanted into the upper end this fragment of tibia from the patient. An autogenetic graft from the patient is by all odds the best. You see the bone has filled clear up to this point I show you. The next picture shows it to greater advantage. When you palpate that, it feels as though you had full and perfect reproduction of bone; but a skiagram never shows young bone early. When a case of fracture of the leg is let out of the hospital, the rule is you can see through the line of fracture all the time while the patient is in the hospital or until he is discharged, but it is united just the same, although the deposit of calcareous material is not sufficient to interrupt the transmission of the x-ray.

6. Here is a case which gave us much worry as to whether the leg would live after we had done an arthroplasty on the knee. This woman had an ankylosis of the knee for thirty years, and the heel was up in this position close to the buttocks [indicating], and this photograph shows the bowing that occurred in the tibia. The ankylosis occurred when she was a girl of seven. Notice the curve that has formed. This is a lateral view at the point of ankylosis. We brought that around, and in doing so we put enormous tension on the popliteal structures. We exposed the arteries and veins and nerves, but all three are now in position. The next few days following the operation she had edema and swelling and cyanosis of the foot, so that I thought the leg would surely die, but it lived. Here is an x-ray picture after the operation (Figs. 45–47). In twenty-four hours motion and sensation had both disappeared on account of the stretching of the popliteal nerves, so that she has now paralysis of all the muscular groups below the knee. Her sensation has already come back. It is only a question of a short time when motion will return. These nerves are not cut. She will get complete restoration of both motion and sensation. I show you the degree of motion she has already. She is going to have splendid motion. She has a little necrosis of the skin, and this we will cover with scarlet-red ointment. She has a perfectly movable patella, and it is only a question of time when she will have a knee flexing to right angles. Remember,

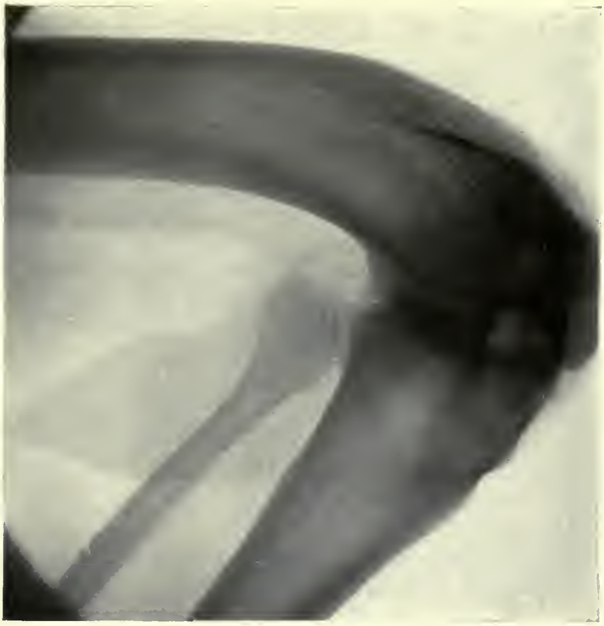


Fig. 45.—Skiagram of Case No. 6 in exhibit of cases, etc., showing ankylosis of knee-joint.

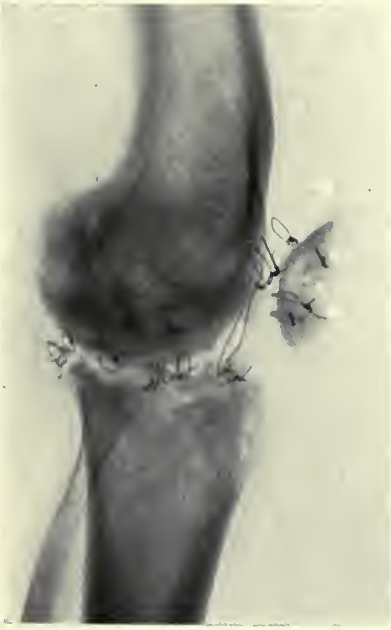


Fig. 46.—Skiagram of Case No. 6 in exhibit of cases, etc., showing results after operation.

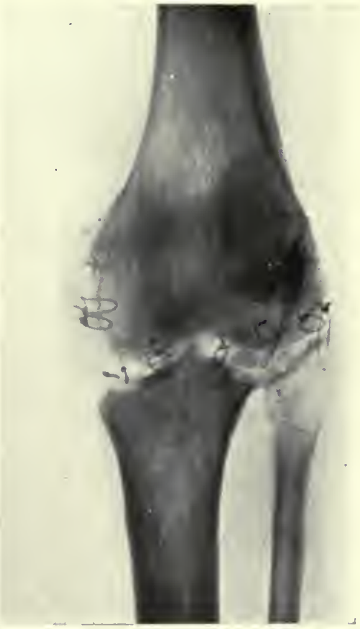


Fig. 47.—Skiagram in Case No. 6 in exhibit of cases, etc., showing results after operation.

this quadriceps muscle has been out of commission for thirty years, and all this will be developed as she commences to exercise.

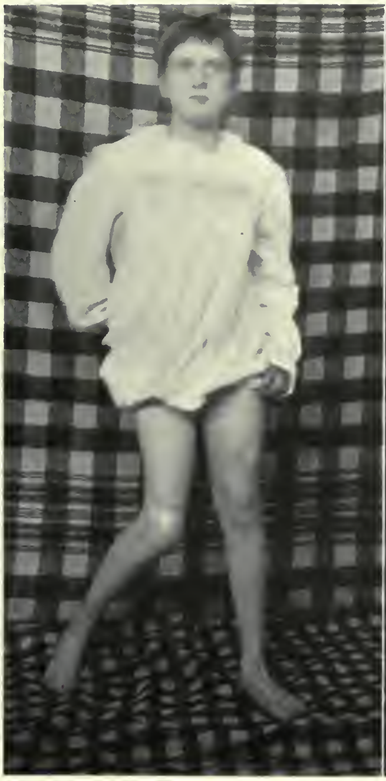
7. The next case is one which involved a new principle, that is, of taking a flap from a distal portion, entirely detaching it, and putting it in between the bones. It was all cicatricial tissue around the knee. We could not get a flap from any place in the neighborhood of the knee to put in, so we took the aponeurosis, that is, the fascia lata, which covers over the trochanter, and implanted it in this case. There was complete bony consolidation, with almost complete absorption of the patella. You see the abduction of the leg. You see the graft. There is complete bony separation, showing a splendid result. You could not tell this from the normal knee except for the little exudate and wire around the side and the smaller patella (Figs. 48–52).

That settles the principle of fascia transplantation from a distal portion. We have completed the last link in arthroplasty. Of course, variations in technic will be made, but the principle is established. We do not claim much, but we do claim credit for establishing this principle. The field for arthroplasty is enormous; in fact, one is surprised at the vast field there is for this class of work. It is very much like appendicitis. In the early days, when appendicitis was brought to the notice of the profession, I remember reading a paper before the Chicago Medical Society on that subject when the dean of the medical profession of Chicago, who was president of one of the first-class medical colleges in Chicago, not according to Flexner, but still true, and I reported eight cases of appendicitis in that paper in 1889. He said, "How is it that a young man, who has only been in practice a short time, has had eight cases of appendicitis (perityphlitis) in a few months, while I have been engaged in active practice for over twenty-five years and have not seen half of that number?" It simply means he could not see or recognize them. They did go to the doctor. And it is just the same with these cases of ankylosis. The woods are full of them, and the field for doing this work is enormous, and it is for the younger men or our older men to perfect themselves in the technic, because the anatomic principle is definitely established for every large joint in the body, and the results are assured if the technic is carried out properly.

The avoidance of infection is the all-important matter in this work, and it is a work in which infection is more difficult to avoid than in the peritoneum, because the tissues have not the power of resistance that the peritoneal tissues have. When infection does take place, however, it is not so dangerous as it is in the peritoneum. We have only had one fatal case. We have had no return in any of the cases we have been able to trace up to date, that is, no return of bony union. We have one in which we believe there is a return of bony union. This was an elbow case. The patient was a woman who was operated on in the early period, and she would not stay in the hospital to have massage because it hurt her. In the fatal case, we operated on one hip of a man with double hip-joint ankylosis. After he was able to leave the hospital he went home and came back three months later for the second operation. He had the morphin habit. We thought we had cured him of this, but on the tenth day, after the second operation, he had a severe pain, pulled the dressings off one night, and the wound was wide open. He got suppuration, and died three weeks later from sepsis. It is the only death we have had in all the cases. In other cases we have had saprophytic infection, but not a pyemic infection and rarely superficial infection.

8. This is a case of double ankylosis of the hip. The patient was ankylosed from his knees up to his second cervical vertebra. When he got to the side of his bed he had to fall into it. Here is a photograph showing both hips ankylosed in the position you see. Here is another photograph after the first operation, and here is still another after the second operation. I want to show you the line of incision. You will observe that he has perfect freedom of motion. See what a perfect joint he has. Here is the other one that has just been operated on. This patient works his limbs day and night—so much so that I believe he moves his legs in his sleep. It was eight weeks ago since the last operation was done. This man will be a producer now instead of a burden to society. He will be able to walk without crutches and without a cane.

9. In this clinic we are compelled to have "befores and afters."



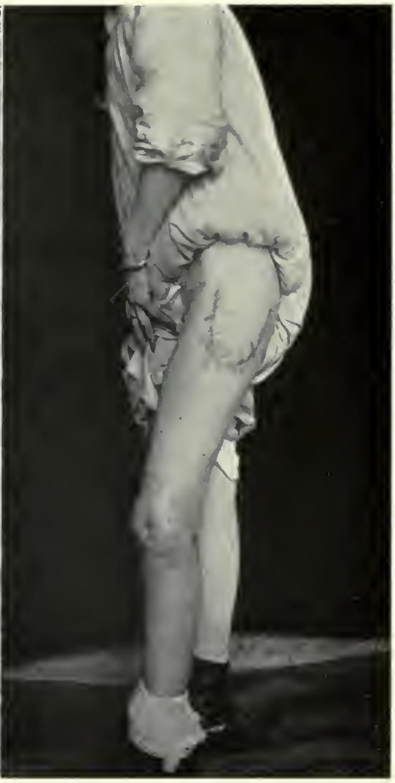


Fig. 48.—Photograph of Case No. 7 of exhibit of cases, etc. Condition and abduction before operation.

Fig. 49.—After operation, showing where the fascia lata was taken for flap.

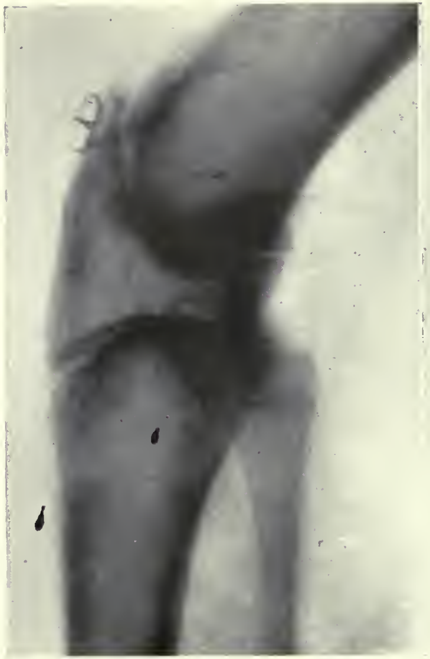


Fig. 50.—Skiagram in Case No. 7 in exhibit of cases, etc. Ankylosed knee that had previously been operated on. *X*-ray before operation, showing complete ankylosis.

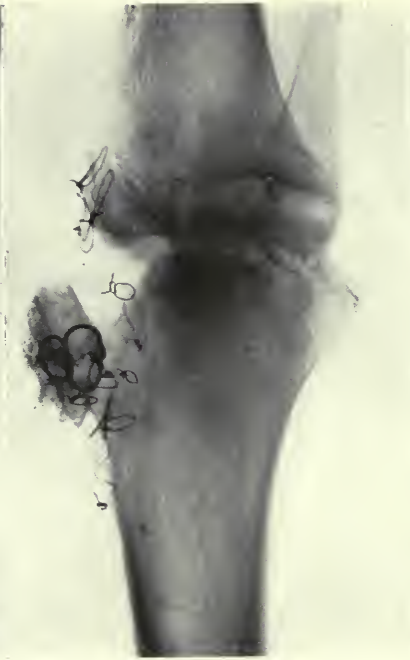


Fig. 51.—Skiagram in Case 7 after operation.

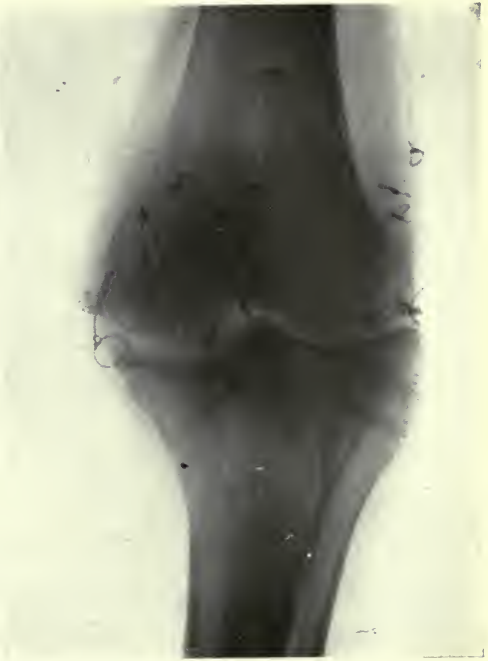
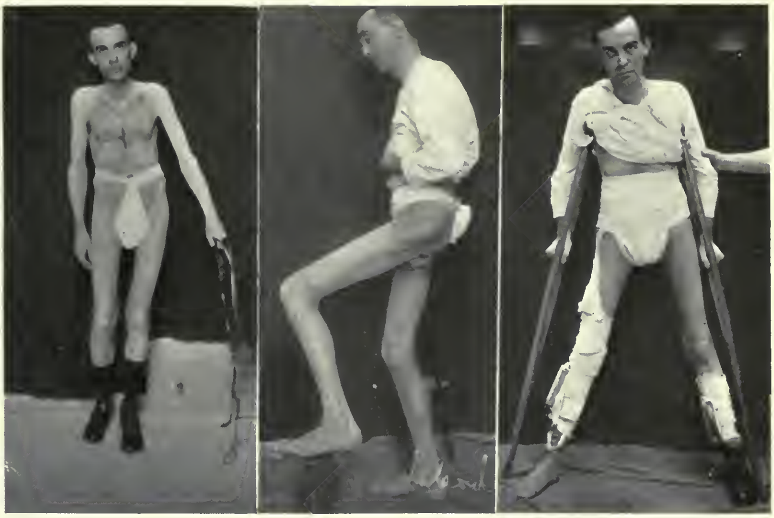


Fig. 52.—Skiagram in Case 7 after operation.



Before operation. After first operation. After second operation.

Fig. 53.—Photograph of Case No. 8 in exhibit of cases, etc.

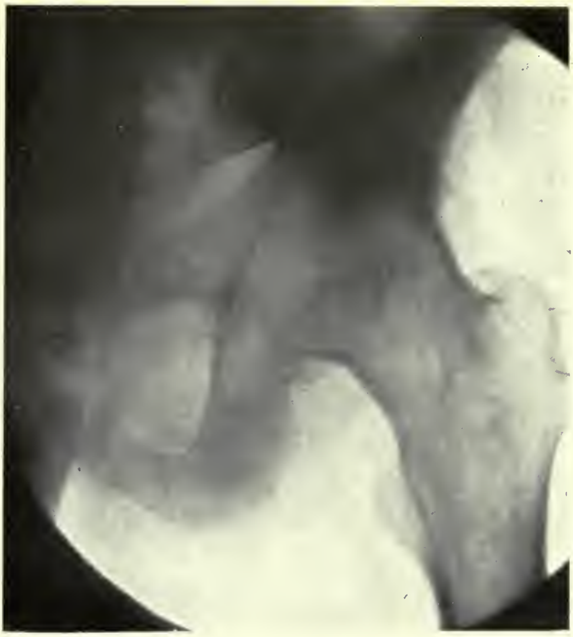


Fig. 54.—Skiagram of Case No. 8 in exhibit of cases, etc. Showing one hip after operation.

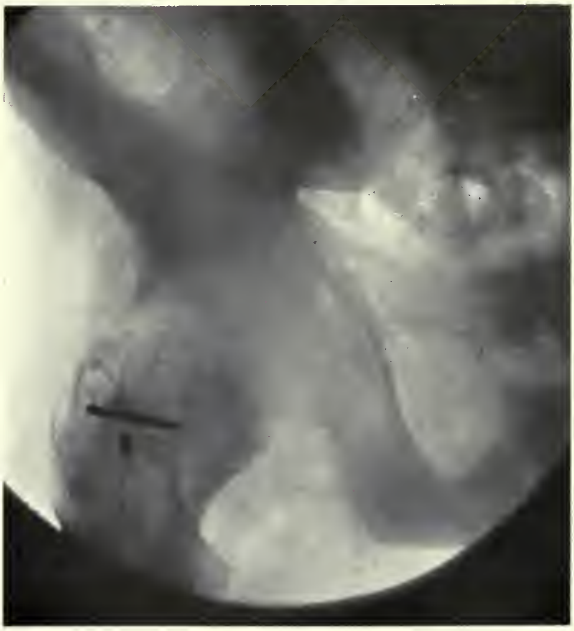


Fig. 55.—Skiagram of Case No. 8 in exhibit of cases, etc. Showing bony ankylosis before operation. Both hips are practically the same.

This is one of the "afters." We operated on this man on the tenth day of November, 1909, for duodenal ulcer. It was one of four cases of duodenal ulcer from the same town which came in in three months. The town has a population of only 2800. The ulcer was easily demonstrable at each of the four operations. The ulcers were all diagnosed and correctly by the local doctor, much to his credit. When this man presented himself for operation he weighed 162 pounds. He now weighs 230 pounds. He said that he had been feeling bad for from fifteen to eighteen years before he was operated on. This is one phase of the results in operation for duodenal ulcer.

Unfortunately, it has another side. It is not all sunshine in these cases. The older the ulcer, the larger the ulcer, the more severe the lesion, the greater the relief and the longer the relief continues. That is a peculiar thing. It is just like the removal of the kidney. If you take out a kidney that has only a little of its tissue destroyed, you hazard the life of the patient 30 per cent. —that is, 30 in 100 die. If you take out a kidney, of which little is left,—only a shell,—you hazard the patient about 2 per cent. It is exactly the same with these cases of duodenal ulcer. If you have a severe old lesion with cicatricial contraction and do a gastro-enterostomy, as was done in this case, you get a perfect result, and these patients go around shouting your praises. If you have one with a small ulcer outside of the pylorus, or a small ulcer just inside the pylorus, and you do a gastro-enterostomy, in a considerable percentage of them you have distressing symptoms at a later period, and the question that is going to be settled is right up to the operators now, namely, that in cases in which there are extensive ulcerations and adhesions there is no indication for excision; in the first place, it cannot be done; in the second place, you do not need to do it because these cases get well after gastro-enterostomy. The *little ulcers*, those just inside or outside of the pylorus, are the ones we are going to excise. We shall then close up the opening and do a gastro-enterostomy, and then I believe we will get better results.

I had in my office day before yesterday a physician who had an ulcer of the duodenum. He was operated upon a year ago last October. For six or eight weeks he had perfect relief. Now he has exactly the same symptoms that he formerly had. He has food distress; he turns over on his side in bed and pours the food out of his stomach, and he has all the symptoms that recur following a gastro-enterostomy. The probabilities are that he will have to undergo another operation. I did not operate upon him originally. In his case I will either have to do an entero-enterostomy or a Roux operation, or with an hour-glass pylorus do probably both at the same time. He has what he calls a typical vicious circle, and I think he is right, and still it was a no-loop operation. We are all talking about and endeavoring to avoid a vicious circle. There is something about it we do not understand, and we may as well stand up and look fate in the face, because there are some things about these ulcers that we do not know and we cannot explain. Notwithstanding that, the severe cases and the late cases give splendid results. An operation can be done with little or no risk to the patient, and when operation does not afford relief, we should go right in and do whichever one of the secondary operations is indicated after the primary operation of gastro-enterostomy. We do not do the entero-enterostomy of Roux as a primary procedure. We do four cases out of five with the oblong button, because we believe in the average case it is the safest and quickest method, but there are conditions that require us to use the suture with the clamp.

10. Here is a case of surgery we are proud of—Miss X, twenty-two years of age. It was a fierce operation, but the result has justified or warranted the effort. This patient scarcely had any courage left for us to do a palate operation, because it was declined so frequently by other operators, but being a young woman, I felt that nothing was to be lost, and everything was to be gained. She had no horizontal palatal plate. There was a perpendicular bony mass, smooth on each side of the naso-oral space, running up to the turbinated bones. The gap between it was a little wider than my thumb. She had a prosthetic mass of hard rubber that went in between the perpendicular plates in the superior maxilla clear back and pressed against the posterior wall of the pharynx, so that she could talk. It was part of a dental apparatus to give

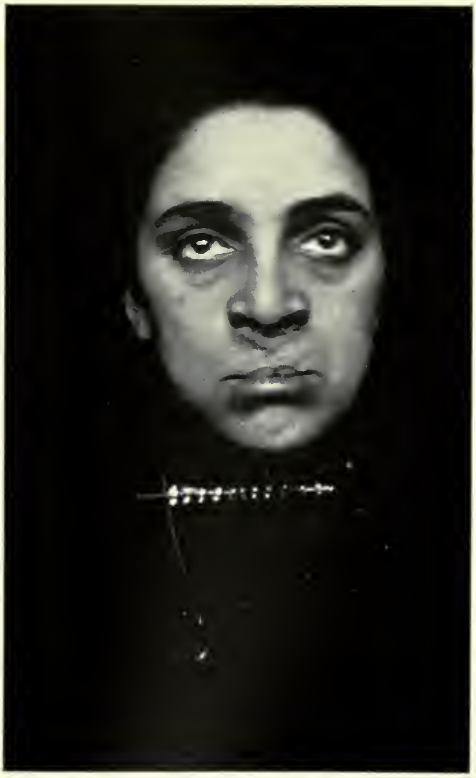


Fig. 56.—Photograph of Case No. 10 in exhibition of cases, showing results of operations.

her an opportunity to speak. We fracture the perpendicular plates of the deformed bony palates with a reverse chisel close to their basal attachment, and bring them toward the midline, making overlapping flaps of the soft palate. We succeeded admirably and have a good hard and soft palate notwithstanding her age. The operation was done four weeks ago. In addition to that, there had been an effort made to close the lip, and it was closed at the angle of the nose, and one could put the index-finger between the two portions of the lip. We operated on her lip a week and a half ago. You can see the plan which we followed. We cut the lip in the manner I show you, swung it round, and took this portion of the nose-flap and spread it across, making a perfect lip, as you see. Look at the thickness of it. This patient has a splendid result. When her teeth are put back she will be able to speak and swallow and do everything that other people do. The lip is full length and full thickness of the normal lip and will have the same mobility.

IMPACTED COLLES' FRACTURE

HISTORY

This patient is a teacher, aged twenty-one. Entered hospital for deformity following fracture of left wrist five weeks after date of accident.

History of Accident.—While patient was walking on the ice she fell forward, catching her weight on the extended left hand. When she arose it hung limp. The left wrist was enlarged, and a prominence existed just above the joint. It rapidly became swollen. She held the hand flexed at the wrist and slightly abducted. A doctor told her it was only sprained, and put on adhesive strips. She wore these two weeks. The deformity still persisted, and she went to another doctor, who put the arm and hand in a wooden splint. She wore this one week, the doctor finally giving up the case, confessing that he could not improve the deformity.

COMMENTS AND OPERATION

This case is a very instructive one, not only from the standpoint of the deformity that is present, but from the misleading character of the skiagram.

If you look at the articular surface of the lower end of the radius in this case, you will find that you are looking onto the face of the articulation. In other words, the face is not square across the lower end, but faces decidedly backward on the lower end. The normal anatomic face of the articulation of the radius is just a little forward. That should always be in the latter position after a corrected Colles' fracture.

In this girl's case there was impaction in the Colles' fracture, and the articular surface faces backward instead of forward. That accounts for the great deformity. When you look at the posterior margin of the radius, you will see there is practically no displacement at all—there is simply an impaction. With impaction there is a change in the direction of the articular surface, with practically no luxation.

The history accentuates what I have said in this clinic so frequently that the most common deformity following fractures in the body is that which occurs in connection with a Colles' fracture. The reason for this deformity is not because the doctor put on an improper splint, but because reduction was not effected. It is one of the most difficult of all fractures to reduce. What is the cause of the deformity from a Colles' fracture? Colles' fracture never occurs from falling on the back of the hand, but always from falling on the palm of the hand. This fractures the radius either straight across or upward and backward at varied angles. The displacement is upward or backward of the lower fragment, with rotation or luxation of the lower end; the lower fragment is tilted backward, or there is displacement of the entire lower fragment onto the dorsum of the upper fragment.

A common method of reduction is to grasp the hand, extend it, and swing it forward. It is never fully reduced by that method. The principle is wrong, and the result is a failure. The only way the fracture can be reduced properly is to increase the deformity, unlock the fragments, push the lower fragment down, and then flex it forward. Put the distal thumb on the posterior surface of the distal fragment, and put your proximal thumb of the other hand on the nail of the first thumb, and increase the deformity in the manner I show you [illustrating on an interne]. I can break that bone if I wish. I could break it easily. If the fracture is properly reduced, the fragments remain in place without any splint at all. The reduction should never be attempted without an anesthetic. It is too painful a procedure. The patient does not let you do your work properly without an anesthetic, and the result is a failure. After it is reduced, apply a plaster-of-Paris splint along the posterior surface of the arm, applying the strips parallel to the long axis of the arm, and incircling the wrist and arm three-fifths of its circumference, or put a roller bandage on it and apply any splint you wish. The arm can be carried in a sling after reduction has been properly effected. It does not make a particle of difference whose splint you use if the

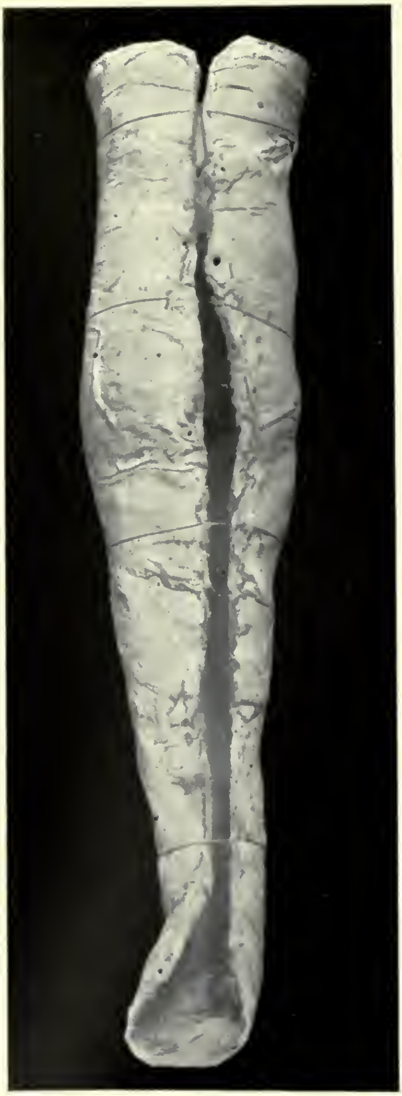


Fig. 57.—Plaster cast used for the leg, showing the manner in which the cast is cut with the Gigli saw to allow for swelling and edema of the limb. The same plan is adopted in casts used for the arm and in other portions of the body.

reduction is properly brought about. It does not need a tight retention bandage. If the patient does not sleep the night after the fracture, then your reduction is incomplete. If the patient has pain that keeps him awake, go right back, do the work over again, and make a complete reduction. First increase the deformity by overextending the hand; when the deformity is increased, push the thumb down on the lower fragment, flex it forward, and that is all that is necessary for success. Then let the patient hold the hand up; if it stays perfectly straight, the reduction is all right. If it tilts, reduction is incomplete. Run a strip of plaster-of-Paris up and down the arm, and put a little roller bandage around and keep it still. Let the plaster come up the finger base and tell the patient to use the fingers right away.

This is an annoying deformity. What impairs the usefulness of the hand is that the lower sharp edge of the upper fragment is displaced forward and presses against the flexor tendons. The flexor tendon-sheaths are very sensitive, and pressure by that sharp edge, when the patient uses the hand, is very annoying. Do not keep the wrist bandaged tightly for six or eight weeks trying to get an unreduced fracture straight, because, having committed a primary crime, you commit a secondary one by causing adhesions of the tendons over the sharp edge of the lower fragment, and, as a result, the patient has limitation of motion in addition to deformity. While patients put up with deformity very well, they do not put up with limitation of motion. A useless hand is a very serious matter for a patient.

You will notice in this case that the articular surface is tipped backward. We must divide the bone in a **V** shape and rotate the articular surface forward so as to stop its throwing itself back in that position. It is not to be cut straight across; we will make a little **V** so that we can slip it around and produce the least possible defect.

When you look at this skiagram, without thinking you would say, first, that there was no fracture; second, that possibly that is a splendid result. It is a splendid result if you fail to consider the angle of the articular surface, and I am speaking now from an *x*-ray standpoint.

I want to show you this deformity. See how this fragment is thrown back! This point of bone is pressing against the tendon, giving her pain all the time. This posterior prominence will not be materially altered. The prominence in front will be altered materially. It does not seem possible that that can be the *x*-ray effect of that hand except for the one thing—a change in the articular surface. Here is a typical deformity of a Colles' fracture. I keep looking for that to see whether it is true. It is one of the most difficult situations to figure out. I never saw it before, and would not have recognized it without the assistance of the *x*-ray. When you examine that skiagram you will say that it is an excellent result, and it is, as far as the anterior or posterior replacement of the bone is concerned, except in so far as the restoration of the articular surface is concerned.

We will expose the seat of the fracture by making a longitudinal incision, about four inches long, over the radial side of the wrist, keeping posterior to the extensor longus pollicis, cutting through the skin and fascia until we see the tendons in their sheaths. These are retracted carefully so as not to traumatize them. The bone is now in view.

I am going under the periosteum. The periosteum is thickened. I am not opening the joint. The line of fracture is seen beautifully here. There is quite an impaction. Now, we must reduce the displacement. I am refracturing the bone as near as I can in the same line, and then make the V-shaped incision mentioned. When I complete the incision, I will feel sure that I will be able to rotate the axis of the lower portion around on that upper part without the slightest danger of luxation, either anterior or posterior, and that was the purpose of making the V-shaped incision in the bone. I did nothing with the ulna except to reduce the deformity; the ulna again assumes practically its normal relationship. It is a fact that there is a shortening and internal displacement of the radius that causes the ulna to become so conspicuous. The bone will rapidly fit in that position. It is in a state of incomplete ossification, but it is in a position where rapid regeneration will occur, because the bone in the neighborhood has been prepared for the advancement of the

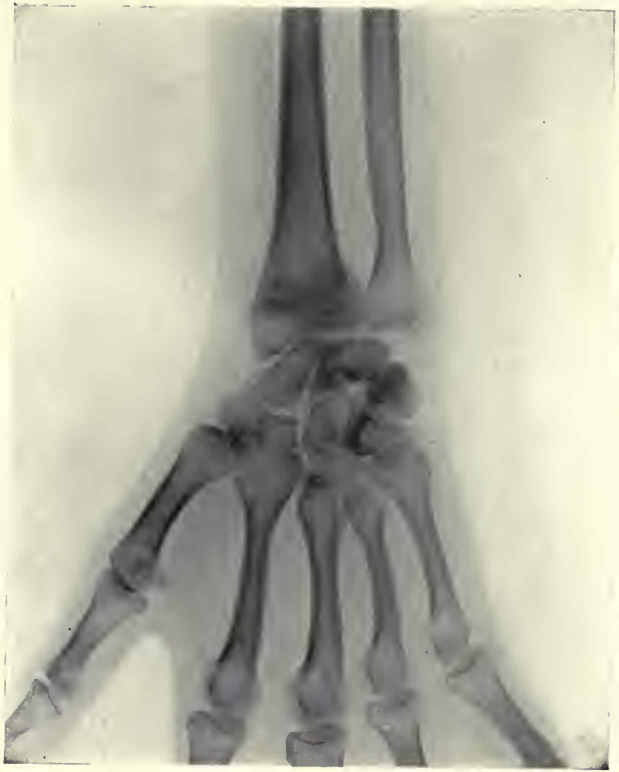


Fig. 58.—Skiagram showing impacted Colles' fracture.

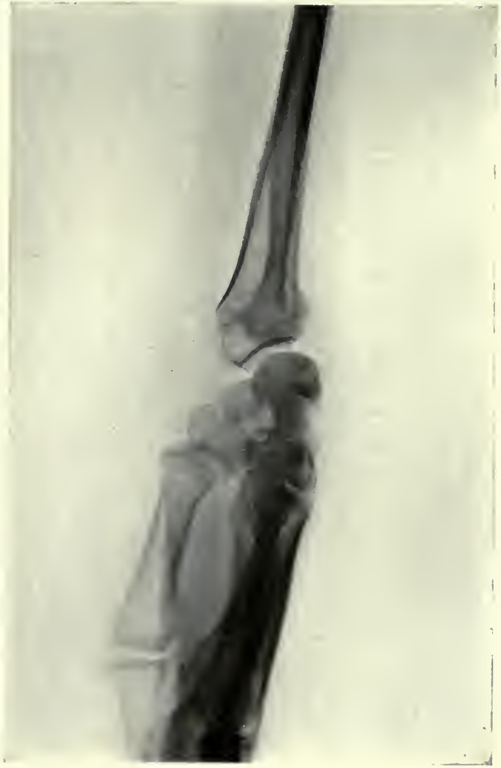


Fig. 59.—Impacted Colles' fracture. Skiagram showing impaction with change of articulating surface upward and backward.

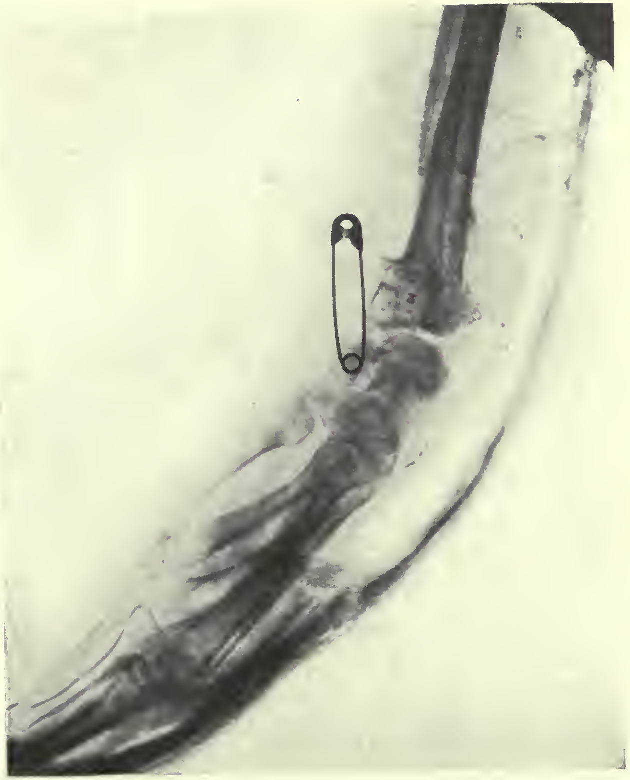


Fig. 60.—Skiagram after operation, showing reduction of deformity and articulating surface in normal position.

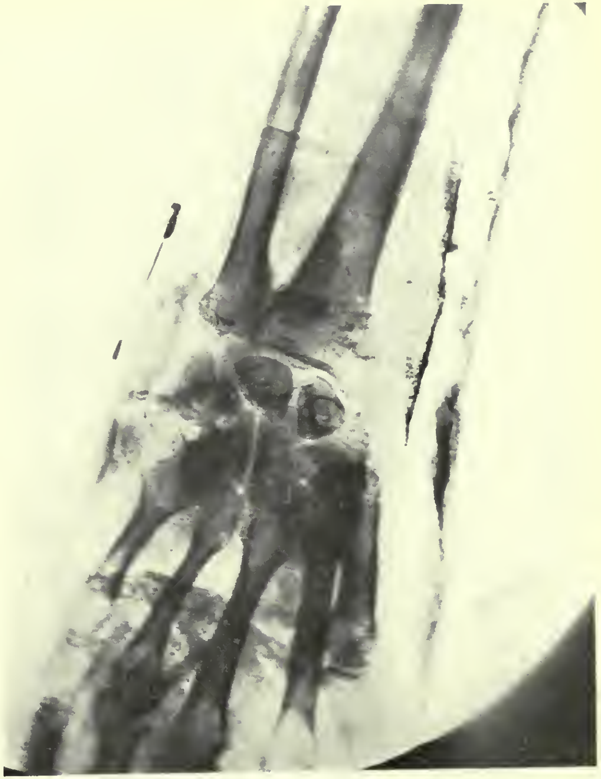


Fig. 61.—Impacted Colles' fracture. Skiagram taken after operation, showing reduction of deformity.

ossifying process. The wound is closed with catgut in the deeper layers, and horsehair on the surface. A circular plaster-of-Paris cast is then applied, in the manner previously described, and as soon as it has set it will be split with the Gigli saw. We do not disturb the fracture in the least by so doing. This makes practically only a partial cast, which is sufficient for retentive or supportive purposes, and at the same time may be spread easily and not cause any pressure paralysis to occur when the arm and hand become edematous. Too tight a cast or even a constricting cotton bandage will cause a Volkmann's or an ischemic paralysis.

FRACTURE OF THE OLECRANON PROCESS

HISTORY

Surgeon, aged fifty, entered hospital February 6, 1912, because of injury to right olecranon process.

Present Trouble.—February 3, 1912, patient was walking on icy sidewalk when he slipped and fell, catching weight on right hand with arm partially flexed. He did not strike on right elbow, but felt something snap, and on getting up found that he could not extend right forearm, and has not been able to do so since that time.

Examination shows tip of olecranon of right ulna separated from ulna and retracted upward ½ inch. Patient can flex arm perfectly, but power to extend it is gone.

COMMENTS AND OPERATION

The history which you have heard tells the story. You see he did not strike the ground with the palm of his hand; if he had, he would have had a Colles' fracture. He came down and struck on his arm so as to put tension on the triceps, and the olecranon snapped off. As you see, with the limb in the extended position, there is but slight displacement. With the limb flexed, there is distinct separation of the fragments. You can easily put the end of your finger in the gap. The question arises as to what to do in this case. In the first place, there exist practically the same conditions as in the fracture of the patella, except that the olecranon has greater osteogenetic power than the patella or than any other bone of the sesamoid group. Therefore, if you secure anything like accurate contact, where there is separation of the fragments you will have bony union. If you should dress the arm in extension and permit these fragments to unite, the upper fragment will be tilted into the olecranon fossa, so that when the patient attempts to extend the arm later complete extension will not be possible. That is one of the dangers. It is a greater danger than the danger of separation with ligamentous union. A ligamentous union in the olecranon is more detrimental to the final usefulness of the arm than a ligamentous union in the patella. The latter gives a fairly good and serviceable joint in many cases.

About two years ago we had in this clinic a patient who fractured his patella in the Civil War. The fragments separated and separated and separated until the gap measured about 5 inches. Still, he had perfect use and perfect function of the limb. He has used it ever since the Civil War as though he had never had a fracture. That accentuates the position we took some years ago, that a patient could get along perfectly well without his patella if he had disease requiring its removal, and that the quadriceps tendon could be utilized as a flap. By splitting the quadriceps tendon upward ½ or ¾ inch from its attachment to the patella in two parallel lines, dividing this section 5 or 5½ inches above, you can turn it down and suture it to the ligamentum patellæ, securing perfect restoration of function. The patient can use the joint as though he never had had any trouble with it.

We have had some beautiful illustrations of that in cases of tuberculosis of the patella where we took out the patella and used that strip of the quadriceps tendon as a support. In our last case of fracture of the patella—a stout man—we used exactly the same means of assisting wiring of the patella. We first wired the patella by the transverse loop plan, split the ligamentum patellæ longitudinally, split the piece from the quadriceps tendon, as described above, turned it down, inserting it into the slit in the patellar ligament, and locked it in there with sutures, and we have not the slightest apprehension about that man's patella. We know that either of these means will give him all the support he needs for the remainder of his life, notwithstanding that he is a very stout man. This is the last fracture of the patella in which we used that plan. In 1904—eight years ago—we operated on a patient and used the quadriceps tendon after the plan I have mentioned, after removing the patella completely. He had tuberculosis of the patella. He has almost full degree of flexion and easily supports his weight on the leg.

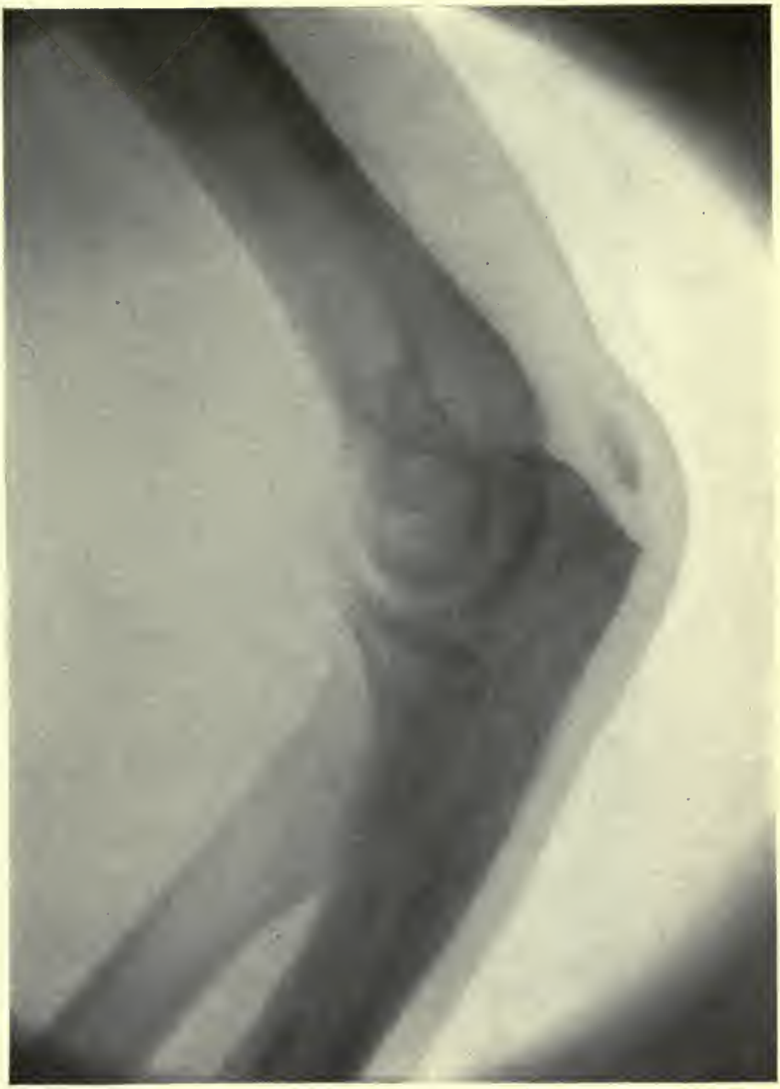


Fig. 62.—Skiagram showing fracture of tip of the olecranon.

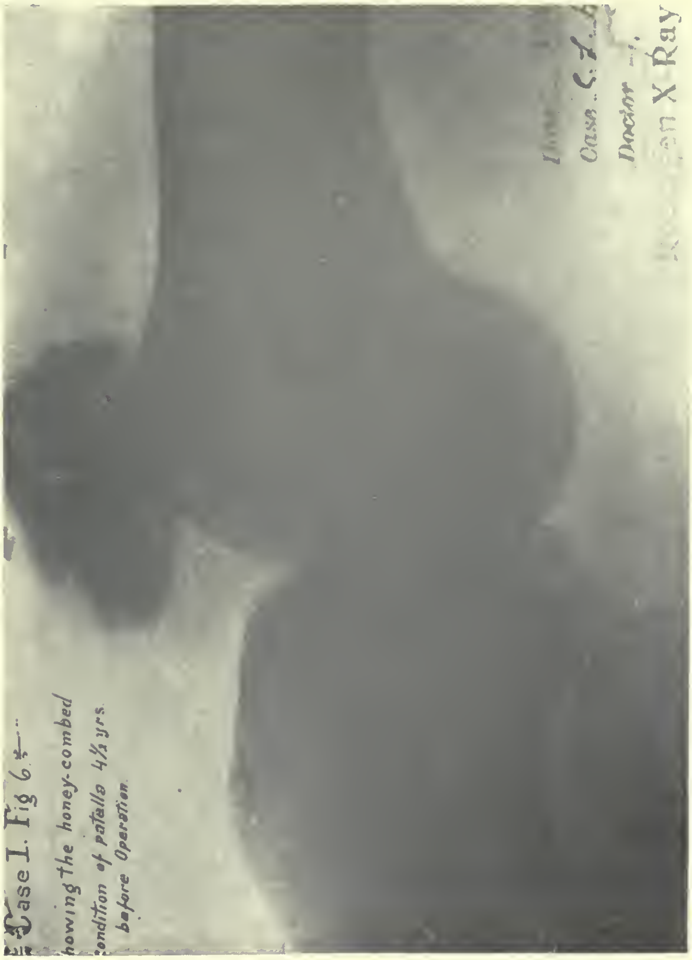


Fig. 63.—Shows condition of the patella, January 22, 1900.

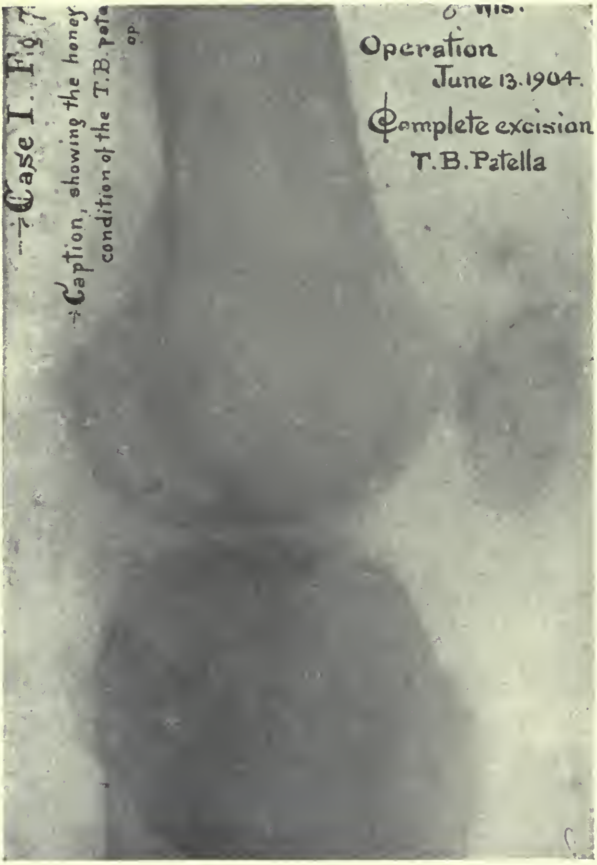


Fig. 64.—Showing the honey-combed condition of the tuberculous patella six months before operation, January, 1904.

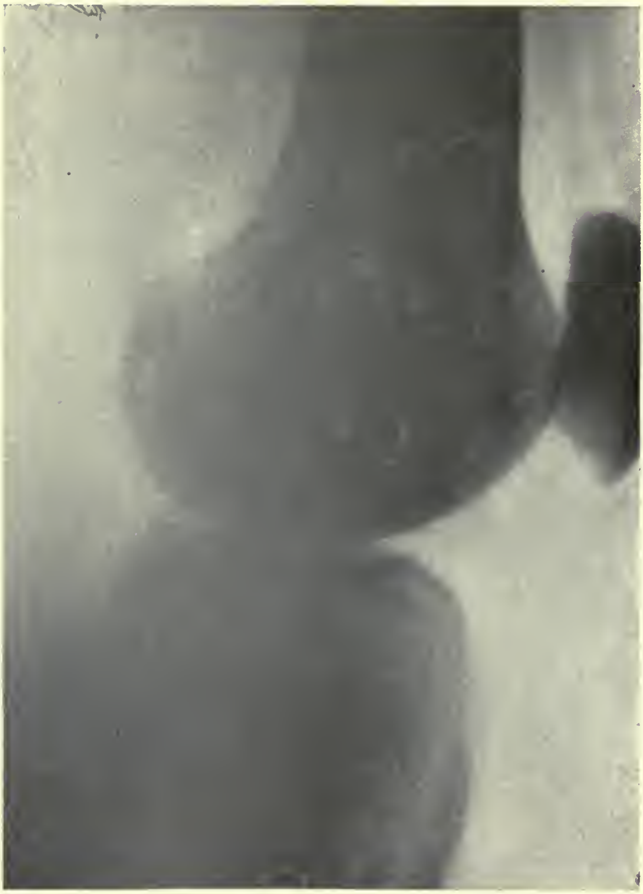


Fig. 65.—Showing normal patella in the same patient.

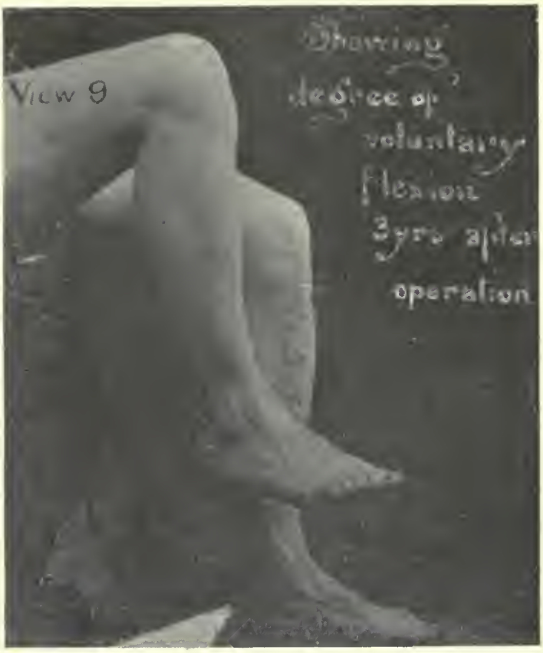


Fig. 66.—Shows the degree of voluntary flexion three years after operation.

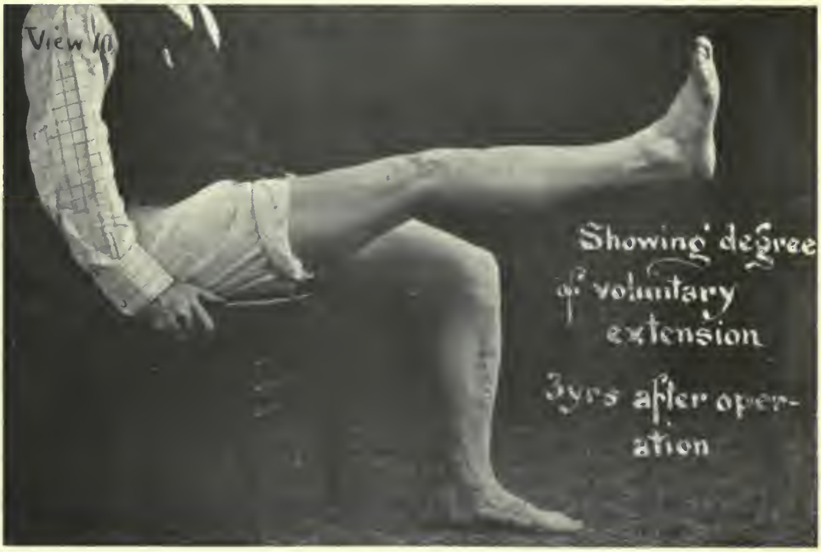


Fig. 67.—Showing the degree of voluntary extension three years after operation.

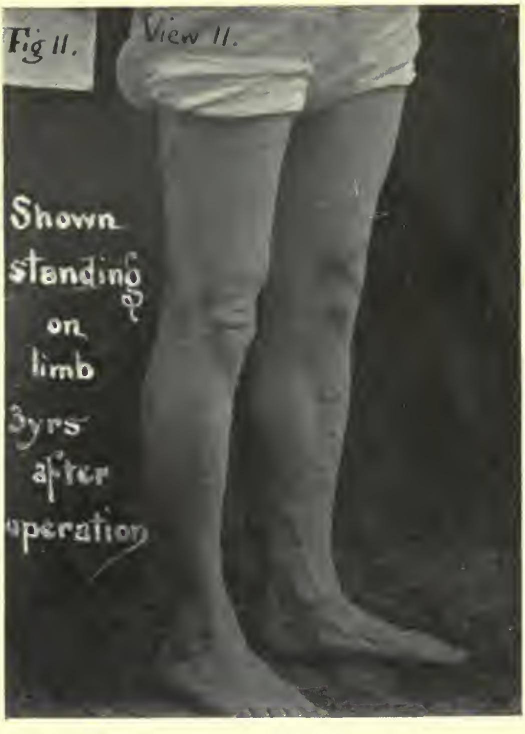


Fig. 68.—Showing patient standing on the limb three years after operation.

In all cases of fracture of the patella where we have the slightest apprehension about the subsequent result after the patella has been approximated by the circular wire loop we support it with a strip taken from the quadriceps tendon, as described. Many years ago I suggested the plan I use now, namely, of looping this patella entirely outside of the joint.

We feared the joint more then than we do now, but we never have ceased to hold a most wholesome respect for it. We never go into a joint when we can avoid doing so.

The plan I am going to use in this case is identical with the method I outlined and published in "Surgery, Gynecology, and Obstetrics" in March, 1908, and that I have used ever since, namely, wiring that button of the olecranon back against the ulna without going into the joint. This is done by making four small nicks or incisions.

The technic of the procedure is to nick the skin on the inner side and just external to the ulnar nerve. I always start from the ulnar nerve side because I can control the angle of entrance of the suture and protect the nerve all the time, and I cannot always control the angle of exit. If the drill turns from outside and you start at the ulnar side, you hazard the ulnar nerve, but, starting from the inner side in this way you never hazard the ulnar nerve. The drill is passed through the inner side through the olecranon ridge of the ulna half an inch below the line of fracture. The drill comes out clear through the skin. The skin is nicked. The drill is threaded with phosphor-bronze wire (No. 12 strand), it is drawn through, and the needle is attached to the distal side, passed under the skin above the olecranon, and on a level with the triceps tendon; it then penetrates the triceps tendon above the button of bone, comes out through a very small nick or incision of the skin, and is again passed under the skin back to the original line of entrance of the wire. The limb is extended and the wire is tied, not twisted, in the regular old-fashioned double knot—the marine knot—which gives the greatest security. We put on three knots, and then turn the edge in. That gives splendid apposition when drawn taut. The wire is buried on the inner side of the surface of the ulna, where it is the least liable to cause irritation. If it is annoying or pricking at all, it can be removed subsequently. It should be kept in ten or twelve weeks for the mechanical support, and to avoid liability of accident, although union takes place long before that. When we originally described the technic and practised it, we found it was one of the easiest of all operations to perform in connection with the ulna, and that it involved no risk to the joint whatever.

Note.—Primary union; good functional result.

In doing the operation in this way there is no danger of exposing the joint to infection at any time, and you have a secure approximation.

In fractures of the olecranon you do not have interposition of the aponeurosis as in fractures of the patella. You cannot do subcutaneous wiring of the patella on that plan with assurance as to results, because when the patella is fractured not infrequently the capsule of the patella is torn above or below the line of bone division, and when it pulls apart, the capsule of the patella folds in between the ends. That is why an operation on the patella has to be an open operation, and why, in the case of the olecranon, we can do a subcutaneous operation.

We use phosphor-bronze wire in all these cases because it is made up of many fine strands. The size of wire we use is what is known as No. 12 strand cable, made up of 12 very fine strands. These strands are so fine that if the wire protrudes through the skin and you cut it off, the skin below that level will heal over, which is not true of any single wire that has a much larger diameter, whether it is made of gold, silver, iron, or phosphor-bronze. It is a fact that the connective-tissue cells are able to encapsulate and care for a finer wire, but they are unable to encapsulate and care for the larger strands. The only cases where we make an exception to that rule is in surgical work in the mouth. In the mouth you can put in a large-sized silver wire, because there we have the greatest type of tissue resistance and of regeneration to be found in any part of the body. We close the wound with deep catgut and superficial horsehair sutures. No drain is used. The wound is dusted with bismuth subiodid powder, and sealed with collodion. The arm is dressed slightly flexed, a plaster-of-Paris cast being applied, but not complete. It is cut with a Gigli saw as soon as it has hardened sufficiently to allow for any swelling or edema that may occur. Passive motion will be begun in about ten days or two weeks. The cast will be removed finally in about two to three weeks. Having been cut, it can be removed and replaced as often as desired. Primary union. Good result.

DIVISION OF THE BRACHIAL PLEXUS

HISTORY

This patient is a laborer, forty-two years old. He entered Mercy Hospital February 5, 1912, because of paralysis of and pain in the left arm, forearm, and hand. There have been no nervous disorders among his relatives. He never has been sick in bed in his life. Five years ago he was struck on the head by a falling monkey-wrench. A doctor told him a slight skull fracture had occurred. He had no headache, was not rendered unconscious, and in two days was feeling as well as ever.

Present Trouble.—October 4, 1911, four months ago, patient, while in a standing position, was struck on the left shoulder and side of neck by a falling timber of great weight. He was rendered unconscious and remained so for ten minutes. A local doctor attended him and found the left clavicle fractured about its middle, but not compounded or displaced badly.

Ever since the accident the entire left arm, forearm, and hand have been paralyzed and are the seat of an excruciating pain. This is growing worse continually. The entire extremity has been anesthetic since the accident, save for an area on the back of the arm which is hyperesthetic. The hand and wrist become cold readily, and if allowed to hang at his side, the hand becomes congested and blue in color. The skin over the trapezius and sternomastoid muscle was anesthetic at first, but sensation has returned gradually. The right pupil has been dilated. The left pupil has been contracted to about normal size ever since the accident. Both react to light and accommodation.

Physical examination by Dr. Murphy. A pressure sensitive zone is present just where the cervical nerves of the left side come out of the intervertebral foramina. It will be necessary to expose the nerve-ends and bring them into apposition with the divided trunks.

All muscles of the arm and forearm are paralyzed, also the pectoralis major and minor muscles, the latissimus dorsi, the subscapularis, infraspinatus, and deltoid. The trapezius and supraspinatus are not involved, neither is the diaphragm. The rhomboidei are paralyzed. A hyperesthetic area exists on back of arm nearly down to the olecranon. It extends outward to the outer margin of the triceps muscle, and within and anterior to the region over the coracobrachialis muscle.

COMMENTS AND OPERATION

The history of a blow on the head is sometimes much more important than the mere fact that a patient was unconscious, or that he had a fracture of the skull. When a patient receives a blow on the head and becomes unconscious, it is a serious condition. The question arises, then, was the patient rendered unconscious? This is much more important than the mere fact that he sustained a fracture. How long was he unconscious after receiving the blow?

If you study the history of traumatic epilepsy, you will find that there is scarcely a single exception to two conditions. One is that a patient who receives a blow on the head, whether it produces a fracture or not, who is not rendered unconscious, does not get epileptic convulsions; while a patient who receives a severe blow on the head and becomes unconscious, whether there is evidence of fracture or not, later in life very frequently gets epilepsy, and the severity of the concussion and injury to the brain is estimated by the primary period of unconsciousness. When a man receives a blow on the head and is dazed for a second or so, and then is ready to go on, it really means unconsciousness. The epileptic phase is the most important phase of every injury to the head. The danger of death from a compound fracture of the skull is not so significant and important to the patient as is the danger of subsequently developing epilepsy from the injury, because these epileptic attacks come on one year, two years, ten years, twenty years, and in one case that I remember twenty-seven years, after the injury.

The question that arises in connection with this type of

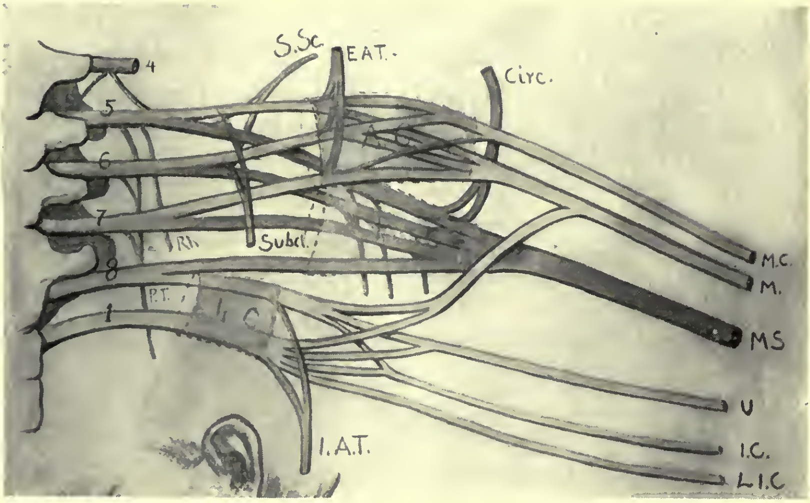


Fig. 69.—Showing brachial plexus.

brachial plexus paralysis has confronted me on four different occasions—the first time when I was a surgeon in the Cook County Hospital. A man was engaged in chopping down a tree. A second tree, which was from 6 to 8 inches in diameter, lodged against the first. When he felled the first tree the second tree fell and caught him on the side of the neck and shoulder, and produced paralysis of his upper extremity, which persisted for three years. Another case which came in later was that of a woman who was in a railroad wreck. Something in the wreck caught her on the side of the neck and caused swelling, infiltration, and edema above the clavicle. While the accident did not fracture her clavicle, she had complete paralysis of all the muscles of the arm and shoulder. The third case was that of a young man who sustained an injury also from a falling tree that caught him in the same way as the first case, with the same result. This patient is the fourth case.

The question arises in this case, Where was the real injury? Has he a division of his brachial trunks as a result of the timber crushing or dividing them, or has he a fracture of his clavicle, with a division of his brachial cords by the fractured bone? Or have the brachial roots been pulled out of the spinal foramina by traction? To reverse that order, then, we will say that if the roots have been pulled out we are not going to be able to accomplish anything. If the injury is a division of the foramina and the trunk ends can be exposed, we hope to be able to get end-to-end union of the nerve, and if that can be accomplished, after removing the neuroma we should get restoration of function. Again, is it the result of fracture of the clavicle, and did the clavicle, when it fractured, compress and cut off the brachial cords by pressure against the first rib? If the latter occurred, then it should have cut off his brachial artery, which it did not do. A pressure that would cut the nerves would surely cut the artery. The nerves, however, come in a little distance outside the artery in that position, and meet in the subclavicular position, though they meet in the upper portion of the axilla.

Again, has he had traction put on his nerves without cutting them, and has he a multiple neuromatous thickening of the trunks

—a much more serious condition than a division? A nerve that has been stretched so that its axis-cylinders are fractured in many places has developed in it a type of connective tissue through which the axis-cylinders do not subsequently pass, and which leaves a permanent paralysis. It usually extends over so long an area that excision with nerve approximation is difficult, if not impossible. It is, therefore, the most serious of all conditions that can occur in connection with an injury to the nerve. It is more common to have that type result from an injury which jerks the arm out and produces extension of the brachial plexus with luxation of the shoulder, than it is with the type that I have just described.

The operation in this case will be a long and tedious process. the question is, What nerves are involved? His phrenic on the left side is not involved. The phrenic nerve is given off principally from the fourth cervical. The fifth cervical is the first trunk of the brachial plexus. He has, in addition, paralysis of his rhomboidei. These are also supplied by the fifth cervical. He has no paralysis of his spinal accessory nerve, which receives its accessory portion from the third and fourth cervical. His trapezius is intact; it is also supplied by the spinal accessory nerve. His deltoid is paralyzed, and the only muscle left of this group which is not paralyzed is the supraspinatus, which is supplied from his fourth cervical.

We will now make an incision extending from the middle of the belly of the left sternocleidomastoid muscle downward and outward, external to the middle of the clavicle, and 3 inches below it. The clavicle is now exposed, and will be sawed through, the ends of the fragments being raised so as to expose the brachial plexus throughout its entire extent. There is a dense mass of adhesions which will necessitate careful dissection of the nerve-trunks. The phrenic nerve is not embedded in the mass. You see it responds to faradic stimulus. After we have separated these nerve-ends from the surrounding cicatricial mass, we will make an anastomosis in the usual manner by splitting the proximal end of the nerve longitudinally and using one of the pieces to bridge over the gap. We will unite the distal portion of the musculospiral nerve to the root of the seventh cervical nerve, and the distal portion of the median nerve to the sixth cervical, using catgut for all sutures, as the retraction is so great that the distal portion of his fifth cervical cannot be approximated to its proximal end at the spinal foramen.

The trunks of the brachial plexus, made up of the fifth cervical and sixth cervical, were cut off just outside the foramen. The trunks of the seventh and eighth cervical were drawn from the roots out of the spinal foramina. The first dorsal was divided just outside of the foramen, leaving the stump on a level with the bone. The sixth cervical was freshened and united end-to-end with the distal portion of the trunk, made up from the seventh and eighth cervical, using very fine catgut sutures. The trunk of the lower portion of the median nerve was split 1½ inches below the point of division in the accident, and we turned that upward to contact with the stump of the sixth cervical. This union was supported mechanically by the scalenus medius muscle. It was impossible to make any contacting of the stumps of the seventh and eighth cervical, and they were taken out, as they had been drawn out from inside the foramen. We have, therefore, in this case the hope of securing permanent axonal contacting between the reflected area of the median and the fifth and sixth cervicals, thus continuing the nerve-current through the median to the outside filaments. This is, to all intents and purposes, a partial side-to-end approximation, which does not, as a rule, give the best results, but it was all that could be done in this particular case. We expect to have a regeneration of the axons of the middle trunk of the brachial plexus. In that way we should get some axonal regeneration in the musculospiral nerve and some in the ulnar nerve, if the suture holds, and there is not too much deposit of connective tissue. The fasciculi of nerve axons were clearly discernible in the distal portion, but were somewhat obscured in the proximal portion, even when it was divided down to the level of the spinal foramen, so that it is questionable how much axonal contacting we will get from the proximal stump of that nerve. It will require from eleven to sixteen months to determine what the final result in this particular case will be.

The clavicular fragments are now united with catgut, and the wound closed with catgut, the skin edges being approximated with horsehair. We will put in a small drain, dust the wound with bismuth subiodid, a desiccating powder, and then apply the dressing.

The result was a primary union. He was examined on May 7th. He has practically no pain, but there is no effort at return of motion, although be believes that the area of sensation has increased.

In treating all these nerve injuries you have to consider these basic principles: First, no matter how long a nerve has been divided, if you recontact the ends without the intervention of connective tissue, you will have a regeneration in the distal axonal portions. We had one case where the nerve was reunited twenty-six years after its division and function was restored. We think that is a good time limit. Second, there must be the absolute contacting of the axons without the intervention of connective tissue, because the axons will not penetrate any considerable depth of connective tissue and establish contact except in the case of the trifacial nerve. In the trifacial nerve there is no distance that the axons will not span. You can cut out 1 or 1½ inches of the infra-orbital nerve, and it will regenerate completely. There is a fairly well-recognized law that nerves have potency of regeneration in proportion to the size of their ganglia. That is why when there are large ganglia on the large spinal roots, as in the brachial plexus, there is great potency of regeneration, and the regenerative potency of the sacral plexus is about in the same proportion. But in the trifacial you have the largest ganglion of any sensory nerve-root in the body, and in that you have a colossal potency of regeneration, not equaled in any other nerves or in any other structure in the body. The tendo Achillis comes nearest to spanning as great a distance as the trifacial nerve and have it fully restored in function. Third, after the division of a nerve-trunk there is always the death of the distal portion of the axis-cylinder, because the axis-cylinder is an elongation of the cell-body of the neuron, and when you separate any portion of the cell-body from its nucleus, the distal portion always dies. In surgery we are acting altogether on the neuron principle, namely, the central cell-body with its nucleus, its axon, its dendrons, and its telodendrites. The dendrons, telodendrites, and axons are elongations of the cell-body itself. The axon is the central conducting wire of the neuron. The myelin sheath is not a portion of the cell-body: it is merely a cement substance, so to speak, for insulating axis-cylinders or axons. The neurilemma is not an elongation of the neuron cell-body; the neurilemma is made up of numerous elongated and elliptic cells contacting and covering the myelin sheath or the axis-cylinder. When you divide an axon, the central conducting wire dies to the periphery, fragments, and is absorbed. The myelin sheath also fragments and is absorbed. The neurilemma, which is the regenerative sheath of the nerve, becomes congested, granular, enlarges by nuclear division, and commences to reproduce a new myelin substance and to produce a new embryonal axon without conducting power. This is accomplished in from eighteen to twenty-three days after division of the nerve. The neurilemma prepares at once the connective-tissue tube through which the axon from the proximal side can pass to the periphery at any time when there is a contacting with the peripheral portion of the axis-cylinder.

It was the belief of Kennedy that there was a new axon regenerated by the neurilemma of the nerve-trunk after the division. I think Kennedy is in error in his theory. The axis-cylinder is not regenerated, but the inclosing connective-tissue tube is regenerated, into which the axis-cylinder passes from the proximal side, and regardless of whether or not Kennedy is theoretically correct, the practical fact is this, that the axis-cylinder or axon never regenerates even to the conducting of faradic electricity until the peripheral or distal segment has been contacted with the proximal living axon. So you see it does not matter whether his theory is right or not, the axon ends must be contacted, and if contacted, the axis-cylinder develops down through the tube which the neurilemma has prepared for it, and develops again to the extreme end, supplying complete motion and sensation over the zone with which it has been contacted.

In estimating the probable result of a nerve suture, then, one has to take into consideration, first, that the ends of the axons are contacted; second, that there will be no intervention of connective tissue between these ends after they have been approximated with suture, and for that reason we embed the line of union either in muscle, which is a desirable medium, as was shown by Levings, of Milwaukee, many years ago, or we wrap it in egg-membrane. If you can, embed the reunited nerve in a muscle, which is most desirable, and sew the muscle all around it. Otherwise, use egg-membrane "as a plumber wraps a joint." This is an absorbable membrane. After you have sutured the nerve-ends together wrap the egg-membrane around the nerve across the line of union. That prevents the intervention of connective tissue from the outside. It is desirable to have a nerve-union produced by the neuroglia type of connective tissue, which permits of the transmission of axis-cylinders better than the ordinary connective-tissue type of union. The nerve-bundles are held together by neuroglia. If you get the ends approximated accurately, the neuroglia forms a definitive union of the nerve, not the connective tissue from the outside, and that is the purpose of wrapping it in muscle or egg-membrane.

There is probably no field of work in which we are at present engaged that is quite so satisfactory as regeneration of the ends of nerves when they are properly united. If you look over the literature of nerve union, you will find that, up to the time we wrote our original article on neurologic surgery, it was divided into two great schools, one claiming that a nerve, if united, will not have its function restored; the other claiming that if the nerve is united, function is restored. Both schools are right, but neither recognized the real key to the success in one or the failure in the other. The reason the men who got bad results failed was because they took the bulb-ends of the divided nerves and united them without cutting off that capsule of connective tissue which insulates the end exactly as a telephone or telegraph cable is insulated. The men who cut off these ends and sewed the brush-like ends of the fasciculi together so as to get contact got good results. Neither, however, knew the reason for their success or failure. The result was that we had the profession divided into two great schools, one saying that nerves would regenerate when united, and the other saying they would not. Both were right, and both were wrong. The nerves would regenerate if the axis-cylinders were approximated and the connective tissue was kept out during the process of repair.

TUBERCULOSIS OF THE INTESTINES—LAPAROTOMY

HISTORY

This patient is a housewife, thirty-eight years old. She entered Mercy Hospital February 20, 1912, because of pains in lower abdomen. There has been no tuberculosis among her relatives. Her mother died of carcinoma of the uterus at the age of sixty-three. Her past life has been free from serious illnesses. Her menses always were perfectly normal until onset of present abdominal trouble, twenty years ago. Since then she has had some dysmenorrhea. Menses always have been rather scanty. A slight leukorrhea began at about the age of eighteen and has persisted ever since.

At about the age of twenty the patient first noticed that abdomen was larger than usual. Enlargement continued and increased steadily during the ensuing five years. Beginning at the same time as did this abdominal enlargement, at her menstrual periods only would she have severe pain in lower abdomen on the left side. It would keep her awake at night and keep her in bed always during menstrual periods. She never has had nausea or emesis, chills or fever, that she knows about. Her general health never was impaired. She had no dragging pains in pelvis and thighs. The abdomen was the size of one six months pregnant.

Three years ago she was operated on. A large ovarian cyst was removed from the left side of the pelvis. The symptoms were relieved by the operation, and her menses continued normal and without the dysmenorrhea.

Six months ago the patient first noticed that at the times of menses the pains in left lower abdomen and pelvis were beginning again. They were never present between menses, and at menstrual periods were exactly of the same nature as those which occurred just previous to her operation three years ago.

Since July, 1911, she has noticed severe pain down in the abdomen, in the median line, at the site of the old scar. This pain occurs irrespective of her menses, and she thinks is of a different nature than the pain on the left side at her menstrual periods. The menstrual pain is much more severe. The menses have not changed in character, amount, or frequency, nor has she ever missed a period.

Examination by Dr. Murphy (*Vaginal*).—A double mass is present—one behind the uterus, which seems encapsulated and movable. It can be lifted up and out of the pelvis. The other is a part of the fundus of the uterus extending up toward and becoming a part of the left rectus muscle. This mass is not at all connected with the other tumor. The mass is hard, runs out to the rectus, and gives some evidence of infiltration into the neighboring structures.

From the physical signs it appears that she should be operated on to remove the deep neoplasm. One cannot say from palpation where the tumor originates. The incision must be on the right side, so as to be to the right of the mass in the rectus muscle.

COMMENTS AND OPERATION

Aside from the statement concerning this fibroid, she had a cyst—an ordinary pedicled cyst—on the left side. What date was the operation?

The Interne: May, 1908.

Dr. Murphy: Now she comes back with a tumor in the abdominal wall. In addition to that she has a small round tumor in the abdomen which I am able to luxate and move upward in the pelvis, but which does not appear to be a part of the round tumor above. The uterus comes up and rests against the under surface of the abdominal wall, and appears to be part of the mass attached to the abdominal wall, although the history of the case does not indicate that there was any fixation of the uterus to the abdominal wall in the previous operation. You can see that it is an extraordinary condition, and when you come to ask yourself, What are the things that this may possibly be? you think, first, that it might be a sarcoma; second, that it is possibly a carcinoma, where there was an adenomatous condition of the ovary, with rupture of an adenomatous cyst of the papillomatous type and infiltration of the wall. Third, it may be a neoplasm of the desmoid type, originating in the muscle of the abdomen. The desmoid tumors, however, are very rare. In all my experience I have seen only one true desmoid that was hard and woody. It feels, except on one side, like a sarcoma. At the first operation the abdomen was closed with catgut. This might be or could be a tuberculoma occurring in the scar of the former tumor, but it feels more like a sarcoma than like anything else. The desmoids are not quite so hard as the sarcomata on palpation.

I am going to open the abdomen through the middle of the rectus muscle. There is no thickening of the layers of the aponeurosis of the rectus at this point, and no infiltration of the muscle itself. We will go toward the midline in the muscle and see whether we encounter infiltration in the muscle as we enlarge our incision. There is a sense of resistance, but it appears to be on the under surface of the muscle, or at the peritoneal margin, rather than in the muscle. It does not appear to infiltrate the muscle. This looks like the properitoneal fat. Notice how cautiously I am going through. There are no adhesions at this particular point. That is fortunate. It is better than I expected. I must inspect every point within before I proceed, because I do not know just where adhesions exist. I am able to palpate and go down on the right side of this mass, and shall endeavor to make an examination of it. I will enlarge this incision upward, so that I may get around the upper margin and palpate it on the right side of the abdomen. Here is the sigmoid above the lesion. The sigmoid runs into the lesion. It is a tubercular ulcer of the intestine, of the sigmoid,—a granuloma,—and adherent to the anterior abdominal wall as far as we are able to make out. That it is a lesion of the sigmoid is easily demonstrable. There are ulceration and thickening of the sigmoid. There is no thickening or ulceration above, and there is no mechanical obstruction. There is some infiltration. In some places on the surface you see normal areas of bowel. Contrast the two.

Should we resect that bowel? No. Formerly that was the plan. Now we do not do it because, when you commence to resect an adherent and fixed tuberculous intestine, unless you can get out all of that tuberculous intestine with a zone entirely free from adhesions in which to make a subsequent anastomosis, it would open again at once, or there would be a fecal leakage. Possibly it is an actinomycosis. I feel a crater in the intestinal mucosa from the other side. The mass in the bowel is probably a tuberculoma. We had one of these cases not very long ago. The patient had a cancer of the bowel with a metastasis in his femur. He came into the hospital on account of pain in his femur.

In this case there is a crater in the sigmoid burrowing forward at a point where it is easily noticeable above the point of fixation. It is my belief that we have here a tubercular ulcer; that it has already perforated the wall of the intestine, and that it is fixed to and has become a part of this granulomatous mass on the anterior abdominal wall.

I saw a case similar to this with Dr. Alexander Wiener some time in August, and I cite that case in order to emphasize the treatment of those cases. His patient had a very large mass over her cecum, pressing against the anterior abdominal wall. An exploration was made, I believe, but I am not positive about that. The mass was found adherent, or possibly it was not. However, that is a matter of no consequence. The result was that this mass filled all the right iliac fossa. The tuberculin test showed that she had a temperature of 103° F. She received 5 to 6 minims of dilution No. 2 of Lucius and Brüning's tuberculin. As to this mass, while it looked very much like a carcinoma, the tuberculin test settled positively the fact that it was not a carcinoma, but a tuberculoma. She has been on tuberculin treatment since August, and I learned from Dr. Wiener the day before yesterday that the tumor which I saw, and which was as large as my two fists, had entirely disappeared. It could no longer be felt in the patient's right side. That is typical of the course in tuberculosis of the intestine under tuberculin treatment.

When you look over the history of cases of tuberculous ulcers of the intestine you will find that it has been divided into two distinct epochs, from a curative standpoint, as well as from a clinical point of view—the pretuberculin period and the post-tuberculin period. In tuberculosis of the intestine and tuberculous peritonitis, when the lesion is circumscribed, the primary indication is to resect the tuberculous intestine. But you must have a peritoneum that is free from tuberculosis when you make that attempt. You must have an intestine that is practically free from adhesions to the anterior or posterior abdominal wall. Why? If the peritoneum is infiltrated with tubercles at the time you do the operation you will, as a result of the operation, frequently have a fistula—a fistula that you will be unable to close.

I recall my first painful experience when I thought I could do better than the masters of their time—Fenger and Senn. A patient came to me at the Alexian Brothers' Hospital with a tuberculous fistula of the intestine. The patient had been operated on half a dozen times. Dr. Fenger had operated on him twice, and, I believe, Dr. Senn had also operated on him twice. I thought I could beat these men on this proposition. I had had a good deal of intestinal experience, and I thought I could follow a line I had laid down and succeed in getting union. I put it together, and put it together, and every time I put it together there was exactly the same result—failure of union and fistulous development. That patient underwent two operative procedures at my hands and went away with a fecal fistula. I learned the lesson that wherever you have the peritoneum involved in tuberculosis, you should be extremely cautious about making any opening in the intestinal tract, because it is easy to open it, but the great difficulty is to get it closed, and that is what you encounter in this class of cases. So here we will make no effort at all at removal. We will first make the tuberculin test, and as soon as she is over the effect of the operation we will continue the use of Koch's old tuberculin as a line of treatment.

You will remember, I just said that where the intestine is free from adhesions we can resect the intestine and establish end-to-end or side-to-side anastomosis, or a button union if the peritoneum is not infected or invaded. If the peritoneum is invaded by the tuberculous process, or has that slimy, gelatinous, cobwebby adhesive surface, do not touch it, because the endothelial cells have been disturbed by the tuberculosis and the intestine will not agglutinate and unite as the intestine should. If you cannot do an excision, what is the next proposition? When you have intestinal obstruction, the next proposition is to side-track the fecal current from the area of obstruction, if it be large. In other words, exclude the portion of bowel that is involved in the tuberculosis. In this case the bowel is involved so low down that, to make exclusion of it, you must establish an artificial anus, and an artificial anus is not justified except where a patient is suffering from the acute symptoms of tuberculous intoxication with a mixed infection; that is, having fever and sweats and all of that. This patient has none of that train of symptoms—none at all, and, therefore, an artificial anus is not justifiable.

The next procedure, when you cannot exclude the intestine, is to put the patient in the best possible condition you can, with the intestine remaining, and without exclusion—which is what we are going to do here, and later, unless the patient commences to improve, if she shows evidences of septic intoxication in connection with the tuberculous infection, then we will make an artificial anus on the left side, above the position where the sigmoid is adherent to the anterior abdominal wall. You can see that this condition is merely a coincidence. The sigmoid has become adherent to the abdominal wall where this patient had a previous laparotomy. She probably had some adhesions there which fixed the sigmoid and caused it to remain in that position, and that is where the ulcer endeavored to penetrate, the ulcerating sigmoid attaching itself to the anterior abdominal wall.

In treating these cases, where we cannot do a surgical procedure without removal of the intestine, where we do not wish to perform bowel exclusion, the next best treatment is internal medication.

In 1891 Paul Goodman exhibited before the Pathologic Society in Berlin six specimens of intestines that had been affected with tuberculosis,—that is, tuberculous ulcers,—in which the ulcers had healed. This occurred in patients who had been treated with Koch's old tuberculin. You will remember Koch's old tuberculin came out the year preceding, and had been used in many hospitals, and used as it was at that time, in very large doses, they had pronounced reactions. Goodman's patient died from tuberculosis of the lung. Virchow, who was the greatest cellular pathologist of that time, said he had never, up to that time, seen a specimen of tuberculous ulcer of the intestine that had fully healed. This threw a new light on the use of tuberculin in tuberculous intestinal lesions. While he had seen lupus heal, he had never seen a tuberculous ulcer of the intestine heal. Then they started in to use tuberculin in cases of ulcer of the intestine. Tuberculin was lauded by the medical press in the treatment of tuberculosis, and was used indiscriminately and injudiciously by the profession; consequently, the results were not so good in many instances as might be expected.

Tuberculin fell into disuse. While it is one of the greatest discoveries of the time, and one of the most potent of medicaments that we have at our command, with the exception of antitoxin and antitetanic serum and the rabies serum, it has never become a popular agent in the treatment of lesions of the intestine, from the fact that it appeared to be such a failure in connection with lesions of the lung. The failure was not due to the drug having no potency, but to the fact that it was given in improper dose. It took about twenty years to learn how to dose it properly. It took twenty years to learn what the opsonins meant, and what the opsonic index meant in the management of disease with the serum treatment. Since then we have used tuberculin judiciously and injudiciously. *It is one of the agents that has to be used with a proper and intelligent knowledge of its purpose, and when this is mastered, it is a very efficient agent.* It is particularly efficient in lesions of the intestines.

Since that time there have been a number of cases of healed intestinal tuberculosis. There are now on record an enormous number of cases of tuberculosis of the peritoneum, both in the male and female, that have been healed with the aid of tuberculin. I do not say that we can *cure* tuberculosis with tuberculin, but that is the word I would use if we can use the word *cure* in connection with medicine at all. In other words, it stimulates the reparative process so that nature is able to heal the lesion. Tuberculous lesions, as you know, in every position the body, are healed by cicatrization; in other words, by encapsulation with connective tissue or by exclusion. In the earlier reports of the use of tuberculin in connection with lupus it was pointed out that it healed by exclusion, by exfoliating, and by throwing off the tuberculous necrotic infected zone, and in that way gave good results. In the intestines it cures in the same way. There is exfoliation of the ulcer surface; then there are cicatrization *plus* cicatricial contraction of that tissue and complete repair. In the process of repair it is not uncommon to have contraction so severe as to produce stenosis of the intestine.

Tuberculosis of the intestine is just like lupus; it can be divided into three distinct classes, depending upon the degree of response on the part of the tissue to the irritation of the infecting microörganism. You will find in lupus, first, a small, circumscribed tubercle encapsulated by connective tissue. First a papule, then a pustule, then an ulcer, then a cicatricial mass, and finally repair. You find in the intestine exactly the same process. You will find in lupus that while the lesion is healing in one place it is breaking down in another. You find exactly the same thing in the intestine. You will find in lupus that occasionally there is an enormous, thickened, infiltrating mass rising half an inch above the surface. That occurs more commonly in colored people, known as the hypertrophic type of lupus. You will find in the intestine the same type of hypertrophic tuberculosis, so that in these tuberculous granulomata in the cecum and in the sigmoid you can find a tumor sometimes as large in diameter as a fist, without perforation. That is the hypertrophic type of tuberculosis. That is, there is an enormous quantity of connective tissue thrown out to defend or prevent invasion of the tissue by the tuberculous microörganism.

Then there is another type,—the circumscribed type,—in which there is a large gangrenous area in the center of the ulcer. That is what you see in lupus. With this type of intestinal lesion you can see as it advances and repairs what happens. The tumor grows in one place, a cicatricial mass develops in another, and finally the cicatricial mass becomes so great that it causes stenosis of the intestine, and later, as the process of repair goes on, there may be intestinal obstruction. The process may be postponed for weeks or years. I had one case in which it is my recollection that eleven years of the disease elapsed before intestinal obstruction was produced. Sixty inches of the small intestine were involved in the tuberculous process. We removed the 60 inches of intestine, which was free from adhesions, and got primary union, and we followed exactly and identically the precautions we mentioned today and carry out in all our cases since that time.

This patient will be given tuberculin. I believe that this is a tuberculoma, and not a sarcoma. The tuberculin will be crowded along, increased in dosage until we get a uniform reaction of 99.4°, 99.6°, and 99.8° F. the next afternoon after she receives the injection. A reaction that elevates the temperature just one degree beyond what is normal in that patient in the afternoon is just the right reaction. It is indicative of the best opsonic index and the best resistance of the patient. The tuberculin will then be pushed and pushed; the general care of the patient will be attended to—she will be urged to lead an outdoor life and all that which goes toward making for the repair of tuberculosis in other positions of the body, and no further operation will be done on her until the time comes when we may have to make a resection of the bowel on account of intestinal obstruction.

This is a most interesting case, because it is the first one of the type that I have ever seen where the tuberculoma attempted to perforate through the anterior abdominal wall in the midline. Usually it comes over the cecum as a large mass with infiltration, or it comes over on the left side just above Poupart's ligament, and you find an adherent tuberculous mass to the sigmoid. In this case it has become fixed to the anterior abdominal wall.

The ability of the intestine to repair itself is great, notwithstanding that it is constantly in the presence of mixed infection. In estimating the destruction of tuberculosis in every position of the body you have to take into account the mixed infection element. Tuberculosis anywhere, except in the meninges, is practically never fatal, except when there is a mixed infection. Think of it! Therefore it is the mixed infection element in all these cases, whether in the lung or the intestine or on the skin, that plays the fatal rôle. There are different degrees of resistance to tuberculous invasion in the serous tissues of the body. The acme of resistance to tuberculosis is in the peritoneum; the minimum, in the meninges, that is, so far as serous surfaces are concerned, without the use of tuberculin the minimum of complete repair is in the intestine. The mucosa of the bladder will repair itself. The mucosa of the stomach will repair itself, but the mucosa of the intestine never repairs itself, according to Virchow, except under the use of tuberculin.

We are going to have about the same contest as to the value of tuberculin that we have had with every other medical agent and with every operation that has been introduced in medicine. Naturally, it is going to be more equally divided in tuberculosis because more than 50 per cent. of the people are going to use it long before they have an intelligent basis for its administration, and it will be given indiscriminately and in improper doses, and the reason for this is that we do not read carefully and we do not act with precision—*precision!* We read an article—get a smattering of its contents. We think we have grasped the author's idea, and we go to work to carry out his idea, when every step of the procedure should be gone over and over again, and, better than all, you should see the author do the operation or administer the treatment, and then try and do it better than he does if you are confident that his results are not up to the standard in his line. Do not try to do it better than he does on the second case. That is what usually happens. It is a confession, but it is true. It is a lack of precision of doing things in a systematic, regular way. It has its disadvantages to the people and its advantages to the profession because it is that willingness to depart from prescribed lines that has made pioneers. But we should not be pioneers until we prepare ourselves for doing pioneer work in a definite line by knowing what has been accomplished in that line up to that time.

Note.—A typical response to the tuberculin test was noted. The patient was placed on the tuberculin treatment, and a report from her physician some six weeks later stated that her condition was improving.

CYSTIC GOITER

HISTORY

This patient is a housewife, thirty-six years old. She entered Mercy Hospital because of an enlargement at the site of the thyroid gland, progressive weakness, and a sudden loss of voice. There has been no tuberculosis, malignancy, syphilis, goiter, or nervous disorders among the relatives. Never before has she had a serious illness. Her menstrual history is perfectly normal. She has borne three children, the last three years ago, since the onset of the present trouble, but the disease seemed to be in no way influenced by this.

Present Trouble.—In 1902 the patient first noticed an enlargement under the middle of the belly of the left sternomastoid muscle. It was not large, and she gave it little thought. During the nine years following its appearance its size increased gradually, but it evinced no symptoms. One year ago its growth seemed more rapid. At times a slight sense of pressure was felt just back of the thyroid notch, and sometimes she would find it necessary to swallow forcibly several times while eating. She has been a nervous woman all her life, more especially so during the past year. She has had headaches and insomnia often during this time. Eight weeks ago the above-named symptoms became more noticeable, and she was no longer able to do her housework. She became fatigued upon the slightest exertion, and general weakness progressed rapidly, though she did not lose in weight. For the past six weeks she has been in bed a large part of the time, mainly on account of weakness. She often has headaches and pains in the back and limbs. Throat has never been sore or inflamed.

March 8, 1912, for no apparent reason, she was seized by a hard coughing spell which lasted four hours, and at its conclusion she found that the power to speak above a whisper was gone.

She had never been markedly dyspneic before, never had coughed much, and never had been hoarse before or since that time. Pulse is 80. Temperature, normal.

Laboratory Findings.—The urinary examination proved negative. The blood examination showed 4,320,000 erythrocytes, 6800 leukocytes, and 90 per cent. of hemoglobin.

Heart Examination.—The heart-sounds were regular. No murmurs were present. The heart was not materially enlarged. A malformation of the radial vessels exists, and this interferes with the estimation of the nature of the pulse, so that another vessel, preferably the carotid, must be used for this purpose.

COMMENTS AND OPERATION

Dr. Murphy: A good history is the thing that tells the story. This history, as it was read, did not tell the story—progressive weakness and loss of voice. It should be progressive weakness and instantaneous loss of voice. What a different thing that is, is it not? Just one word spoils the purpose of the history. She was coughing, and suddenly, during her coughing spell, she lost the use of her vocal cords. That is the point I want to bring out. The increase in the size of the swelling in the neck came that night. Dr. Tivnen examined the vocal cords, and informed us that they are both paralyzed. According to the history, she has exophthalmic goiter, but she has none of the symptoms of Graves' disease, the only one being progressive weakness. She has no tremor; she has no tachycardia; she has no exophthalmos—simply a sense of increasing fatigue, and the paralysis of both vocal cords, which came on suddenly. It came on too suddenly for carcinomatous invasion. How old is this woman?

The Interne: She is thirty-six.

Dr. Murphy: Early for carcinoma, still not at all so early that carcinoma can be excluded. I have under observation at present a patient, twenty-six years of age, who had a portion of the thyroid removed. The specimen was pronounced malignant by a pathologist. You can see why this could be malignant at her age, and if malignant, one of the first things it attacks after penetrating the posterior wall of the capsule of the thyroid is the recurrent laryngeal nerve, and then she could have vocal-cord paralysis. Carcinoma does not begin suddenly, as a rule. Carcinoma rarely ever involves both the vocal cords simultaneously. See the point I am trying to make! The clinical history does not fit the clinical course of carcinoma, because in carcinoma of the thyroid the paralysis is of the slow, progressive type, terminating in a complete paralysis. Dr. Tivnen will tell you about the condition of the vocal cords.

Dr. Richard J. Tivnen.—We are all familiar with Dr. Murphy's insistence that a clear and detailed history be obtained of every case, and the great importance he attributes to the "history" as an aid in arriving at a diagnosis and the formulation of an intelligent treatment.

In the case before us the wisdom of Dr. Murphy's position on this matter is well illustrated. An analysis of this patient's history from a laryngologic point of view presents the following facts:

1. *Previous History.*—Has not previously suffered, and does not at the present time suffer, from any of those diseases which are, more or less frequently, associated with impairment or loss of the voice, such as affections of the nose, pharynx, throat, or respiratory tract, diphtheria, syphilis, scarlatina, rheumatism, typhoid fever, pneumonia, central lesions, cerebrospinal lesions, tabes, hysteria, malignant neoplasms of contiguous structures, aneurysms, or toxic causes, such as poisoning by arsenic, lead, or atropin. She has had a slight goiter for some time past, which has caused her no inconvenience.

2. *The History of the Present Difficulty.*—One week ago last Friday the patient was suddenly attacked with a violent fit of coughing, accompanied by a feeling of strangulation, smothering, and suffocation; a swelling occurred in the neck at the time of the attack, and immediately following the attack she was unable to use her voice.

3. *The Subsequent History.*—Up to the present time she has not recovered the use of her voice; the swelling in the neck has somewhat subsided, and she has experienced no further difficulty in breathing.

Examination of the patient presents the following findings: She exhibits a moderately enlarged thyroid, the right lobe slightly larger than the left; it is uniform, not tender, firm throughout, and moves freely with the larynx; nose, mouth, and pharynx normal; examination of larynx discloses both vocal cords normal in color and presenting no inflammatory infiltration; both cords occupy the cadaveric position; during respiration they are quiescent, and on efforts at phonation they make no attempt to approximate; no evidences discernible of neoplasms within the larynx or trachea; ocular examination discloses absence of exophthalmos or the presence of von Graefe's, Dalrymple's, Stellwag's, or other signs of Graves' disease; tachycardia and tremor are likewise absent; vision, excursion of lids and globe; pupillary reflexes are normal.

The history, the symptoms, the clinical course, and the objective findings in this case lead to the conclusion that the patient's attack of coughing, dyspnea, and strangulation was a manifestation of laryngeal spasm; that the aphonia which followed was brought about by inability of the vocal cords to approximate on efforts at phonation; that this inhibition of vocal function was due to paralysis of the laryngeal nerves which supply the muscles concerned in this act, and, finally, that the entire cycle of disturbance was due to the pressure of the enlarged thyroid upon the laryngeal nerves, resulting in what is known as bilateral paralysis of both recurrent laryngeal nerves, branches of the pneumogastric.

Paralysis of the vocal cords in enlargement of the thyroid gland and similar pathologic conditions are of more frequent occurrence than is generally supposed. The fact that the voice in such affections is frequently not markedly altered is exceedingly misleading, and may cause the presence of a paralysis to remain unsuspected. It is quite possible for a patient to suffer from a moderate paralysis, or a paralysis of a certain group of laryngeal muscles, and at the same time the speaking voice exhibit little alteration in volume or timber. This is explained by the clinical observation that one vocal cord may be paralyzed, and its fellow, during the act of phonation, exhibit a compensa-

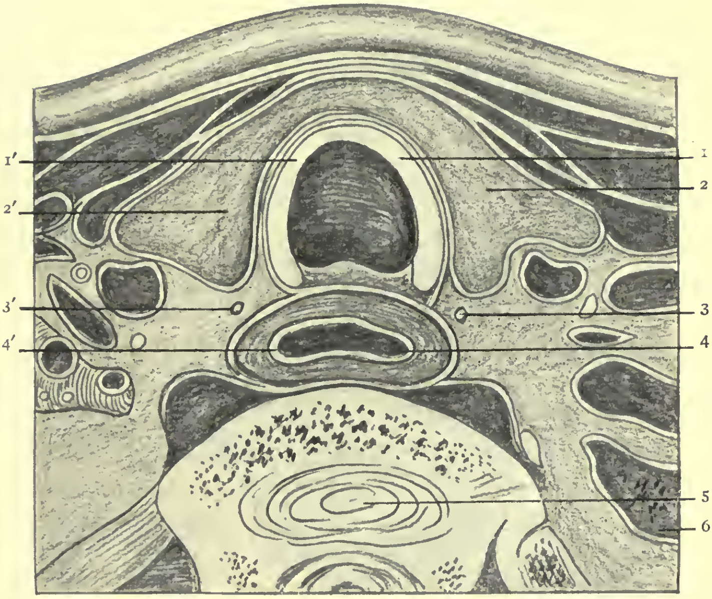


Fig. 70.—Transverse section of the neck at the level of the first dorsal: 1 and 1', Wall of the trachea; 2 and 2', thyroid body; 3 and 3', right and left recurrents; 4 and 4', esophagus; 5, vertebral body; 6, apex of the right lung.

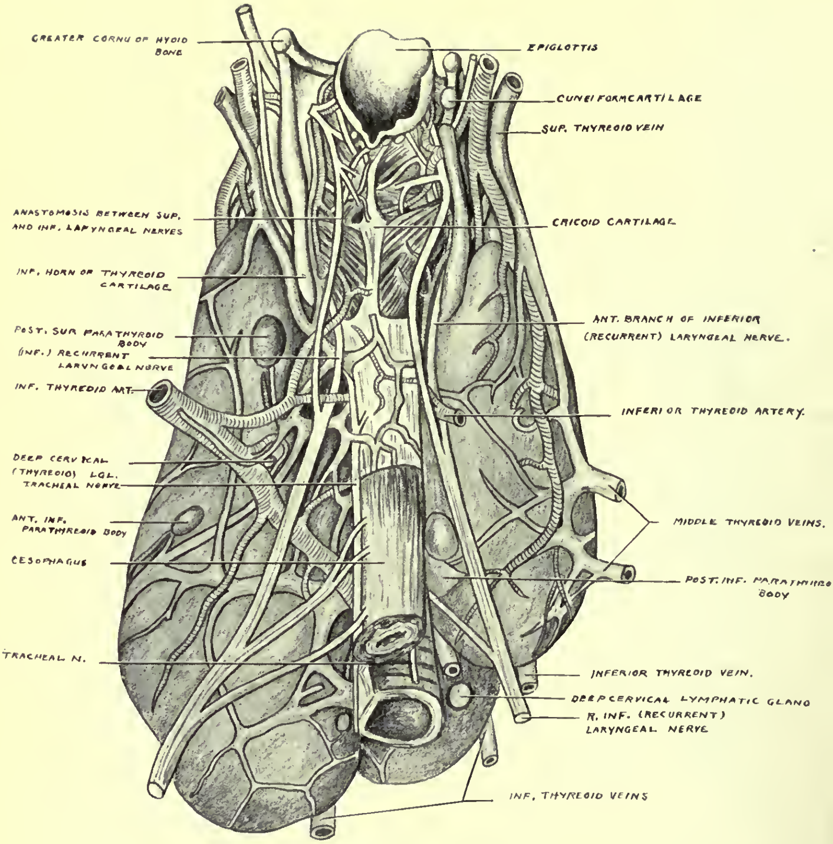

GREATER CORNU OF HYOID BONE
EPIGLOTTIS
CUNEIFORM CARTILAGE
SUP. THYREOID VEIN
ANASTOMOSIS BETWEEN SUP. AND INF. LARYNGEAL NERVES
CRICOID CARTILAGE
INF. HORN OF THYREOID CARTILAGE
ANT. BRANCH OF INFERIOR (RECURRENT) LARYNGEAL NERVE.
POST. SUP. PARATHYREOID BODY
(INF.) RECURRENT LARYNGEAL NERVE
INF. THYREOID ART.
INFERIOR THYREOID ARTERY.
DEEP CERVICAL (THYREOID) LGL.
TRACHEAL NERVE
MIDDLE THYREOID VEINS.
ANT. INF. PARATHYREOID BODY
ŒSOPHAGUS
POST. INF. PARATHYREOI BODY
TRACHEAL N.
INFERIOR THYREOID VEIN.
DEEP CERVICAL LYMPHATIC GLAND
R. INF. (RECURRENT) LARYNGEAL NERVE
INF. THYREOID VEINS

Fig. 71.—Larynx from behind—goiter. (From the body of an old woman the enlarged thyroid gland (goiter) was removed with the larynx and the trachea, and hardened in formalin; only a small portion of the esophagus remains; the posterior wall of the larynx is dissected. The large left lobe of the thyroid gland caused the trachea to rotate around its axis. The recurrent laryngeal nerve has been drawn aside on both sides, in order more clearly to show its branches) (from Bardeleben, Applied Anatomy, Rebman Co.)

tory movement sufficient to cause an approximation of the vocal lips, so that satisfactory phonation is possible.

Matthews, of Rochester, Minn., from an observation on the vocal cords in 1000 cases of goiter, found a total of 289 partial or complete paralyses. The intimate relation of the thyroid to the nerve-supply of the larynx readily explains the vulnerability of the laryngeal structures in affections of the gland.

In Fig. 73 this relation is shown very clearly. The larynx is subcutaneous throughout, while the trachea is so only at its commencement; as it nears the thorax it progressively assumes a deeper level. The thyroid gland, as we see, is situated at the upper portion of the trachea; the base of each lateral lobe extends downward upon the side of the trachea as far as the fifth or sixth tracheal rings; the apex rests upon the ala of the thyroid cartilage. Each lateral lobe averages about 2 inches in length, 1¼ inches in breadth, and ¾ inch in thickness. Occasionally a pointed lobe, the pyramidal, extends upward from the isthmus. The isthmus lies in front of the second, third, and fourth rings of the trachea. The gland is firmly attached to the larynx, and is subject to all its movements; its deep surface is concave and in contact with the trachea, larynx, pharynx, esophagus, inferior thyroid artery, and recurrent laryngeal nerve.

The capsule of the gland is well shown in Fig. 71. The recurrent laryngeal nerves in Fig. 71 are shown in their normal position, external and posterior to the capsule. These nerves are the nerves of the larynx most commonly involved in affections of the thyroid gland. As is well shown in Figs. 70 and 71, they are in intimate relation with the posterior surface of the gland, in a favorable situation to be affected by pressure, and are also a source of hazard in surgical procedures on the thyroid.

In Fig. 72 we can trace the origin and course of the recurrent laryngeals. The left recurrent is the longer, and is given off at the level of the transverse portion of the arch of the aorta, passes under it, and ascends to the larynx. It has an intimate relation to the aortic arch and subclavian; this fact and its longer course explain why it is the most often affected in pathologic processes of neighboring structures.

The right recurrent nerve is given off on the level with the subclavian, around which it curves to ascend to the larynx. Both recurrents ascend in the groove between the trachea and esophagus and end at the cricothyroid articulation by dividing into terminal branches.

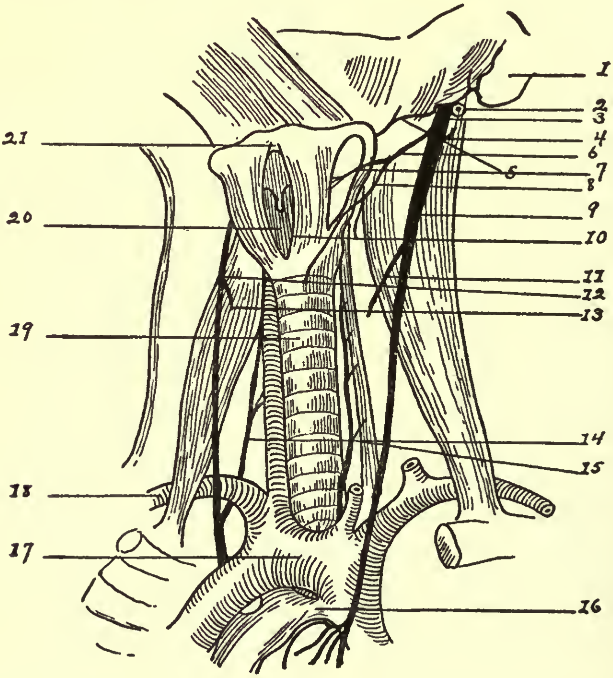


Fig. 72.—Relation of the pneumogastric and laryngeal nerves: 1, Mastoid apophysis; 2, jugular vein; 3, ganglionic plexus of the pneumogastric; 4, internal branch of the spinal; 5, pharyngeal branch of the pneumogastric, anastomosing with the pharyngeal branch of the glossopharyngeal; 6, superior laryngeal nerve; 7 and 8, internal and external branches of the superior laryngeal; 9, left pneumogastric; 10, thyrohyoid muscle; 11, cardiac branch of pneumogastric; 12, right pneumogastric; 13, right cardiac branches; 14 and 15, right and left recurrents; 16, course of the vein; 17, arch of the aorta; 18, right subclavian artery; 19, trachea; 20, thyroid cartilage; 21, hyoid bone (after Ziemssen and Henle).

The external branches of the superior laryngeal nerves enter the muscles so high that only the largest lobes can exert direct pressure on them. The motor impulses of all the intrinsic muscles of the larynx except the cricothyroid are supplied by the re-

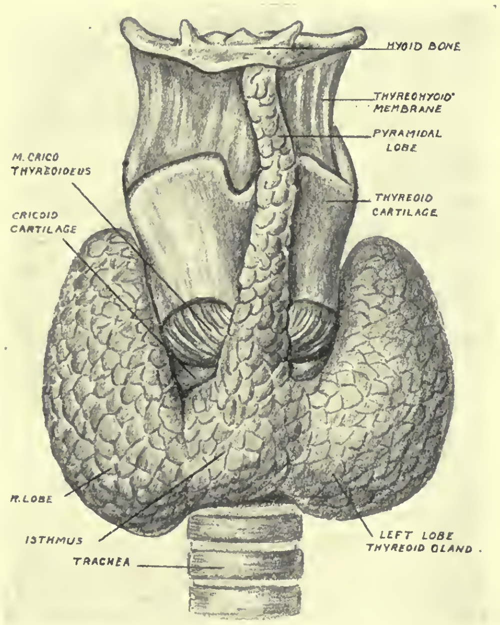


Fig. 73.—Showing relation of the thyroid gland to trachea and larynx (from Atlas of Anatomy, Sobotta-McMurrich).

current laryngeal; it also provides sensory impulses to the trachea and the mucosa of the larynx below the cords.

The superior laryngeal is the motor nerve for one intrinsic muscle of the larynx, the cricothyroid, the tensor of the cords, which modulates the voice; it also supplies the motor impulse to the depressors of the epiglottis, and the sensory impulse for the parts above the vocal cords.

The mechanism of the laryngeal muscles during phonation is exceedingly complicated. Briefly, however, we may distinguish three main movements: (1) Tension of the cords; (2) adduction of the cords; (3) abduction of the cords.

In regard to the character and frequency of the paralyses encountered in goiter cases, Matthews' observations, previously alluded to, are interesting. In his 289 cases of paralysis he noted —"total paralysis of the right cord occurred 18 times; of the left, 30 times. Partial or complete abductor paralysis of the right occurred 50 times; of the left, 83 times. Partial loss of both motions of the right occurred 38 times; of the left, 62 times. Bilateral tensor paralysis occurred in 4 cases."

Notwithstanding the fact that the recurrent laryngeal nerve supplies both the abductors and the adductors of the larynx, clinical observation demonstrates that the abductors are usually the first to become affected as a result of pressure on this nerve. This is known as Semon's law.

The symptoms of compression of the laryngeal nerves vary greatly, and in a general sense may be said to depend upon the size, degree, direction, character, location, and persistence of the disturbing element.

As these factors vary, making pressure on the several adjacent structures,—involving nerve-fibers singly, in groups, or the entire nerve-trunk,—so, too, may the symptoms vary and range from no perceptible voice alteration to complete aphonia; from no difficulty experienced in swallowing to grave dysphagia, and from entire absence of respiratory difficulty to extreme dyspnea requiring tracheotomy.

Matthews found that there was an increase of the paretic conditions as the size of the lateral lobes of the thyroid increased, but that even great enlargement of the medium lobe alone was seldom associated with affections of the cord. This is, as might be expected, when the intimate relation of the lateral lobes to the recurrents is recalled and compared with the absence of this close relationship which the isthmus exhibits.

The position of the lobes, he also found, was equally important; for instance, in goiters of the same size he observed cord affections much more frequent in those cases with lobes located near the clavicle, especially if all or a portion of the goiter is intrathoracic. Hard lobes, likewise, are more apt to injure the nerves than larger soft ones.

The course of the paralyses is influenced by the vagaries of the etiologic factor. The sequence of events in compression lesions is, at the outset, phenomena of excitation and contraction, more or less transitory (spasmodic cough, aphonia, etc.); next, as the nerve filaments are more compressed, progressive motor paralyses as well as atrophy of the muscles innervated by the affected nerve, take place.

The history of Dr. Murphy's case and the clinical findings she presents emphasizes the value of a laryngoscopic examination. As the case well illustrates, the involvement of the vocal cords, due to the pressure of the thyroid, may be a deciding factor in instituting an operative procedure to remove this pressure. The prognosis in the case before us as to the restoration of vocal function is good, in view of Dr. Murphy's decision to remove the goiter—the source of irritation from which she suffers.

As a routine measure it cannot be too strongly urged that the larynx should always be interrogated in cases of goiter. In this connection it is not irrelevant to emphasize that, while the size of the goiter usually bears a direct ratio to the degree and severity of the laryngeal involvement, yet it must be kept in view that the direction, character, persistence, and location of the pressure are equally important, and that, not infrequently, an apparently small goiter, viewed externally, may be the unsuspected cause of irritations to the laryngeal nerves.

This latter type of what might properly be termed cases of "small-externally" goiter may, in their incipient periods of development, for a considerable time, cause periodic, or more or less constant, manifestations of laryngeal irritation, such as chronic cough, hoarseness, etc., and these symptoms be explained on the basis that the patient is a victim of colds or laryngitis. A case under observation at the present time is of this character. A laryngoscopic examination may, moreover, by disclosing an unsuspected beginning involvement of the vocal cords, explain a later postoperative paralysis, thought to be due to the operative procedure, and, in addition, may prove the determining factor in instituting operative interference before distressing or irreparable damage has developed.

DR. MURPHY: With that history, you can exclude acute inflammatory conditions that occur in the larynx, as the patient states that she had no evidence of infection. The question that arises is, Was the cough the cause of the paralysis, or was the paralysis the cause of the cough? Did she have a lesion involving her vocal cords, such as a hemorrhage, or was that irritation of the recurrent laryngeal nerve responsible for her cough? or, when she coughed severely, did it cause hemorrhage into the capsule or into the gland, or whatever the condition may be that produced it, and was it a sequence or an etiologic factor? That is impossible for me to say, and it will not be possible for me to say even after I have made the dissection.

We will make the usual collar skin incision, cutting through the superficial and deep fascia and the platysma. I am elevating the platysma so as to expose the sternohyoid group of muscles. There is none of the usual hemorrhage which one sees in the exophthalmic goiter cases. There is none of that hemorrhage which we see coming from the skin incision in cases where we ligate the superior pole of the gland. The sternohyoid group of muscles is next separated in the midline and divided laterally. These muscles are exceptionally well developed. Their bellies stand out just as shown in an anatomic plate. Elevate them and divide them transversely in such a manner that they will be in readiness for suture subsequently. We must use every precaution here so as not to endanger the recurrent laryngeal nerve. The gland itself is now in the field. It is enlarged considerably, and appears to be quite adherent to the surrounding structures. The vessels in the true capsule of the gland stand out very plainly. The most essential and important thing when operating on a goiter is to have plenty of artery forceps, and you want them in the right place at the right time.

I will free this gland from the outer side, so as to roll it up on the trachea. I am merely steadying the muscles. That allows the gland to come forward easily. With the dissecting scissors of Mayo we set free all the neighboring structures to which the gland has become adherent. The upper pole of the gland passes away around in the neighborhood of the esophagus. The gland is very much larger on the posterior border than one would anticipate from looking at it from the front. The bar or isthmus is also enlarged to such a degree that it was pressing on the trachea. The posterior lobe is larger than all the anterior portion. I have loosened it so that I can bring it forward. We are not removing the capsule of the gland, but enucleating it from behind forward. There is the superior thyroid artery turning upward as it came around from the inner side. There is a parathyroid. I will wipe that off from this capsule so as to be sure that it is not disturbed, and we will commence to go down to the posterior inferior portion of gland that is embedded deep. I expose the artery on the under side. There is the vein. It has a minus pressure on inspiration. You will doubtless remember that some years ago Crile suggested that we leave a portion of the thyroid on the posterior capsule, so as to be sure that we have not cut or injured the recurrent laryngeal nerve. I will peel this gland out of the capsule at the lower angle. The bar that projected down under the sternum is now visible, also the trachea. It is coming out nicely. Here is the isthmus that passes across the trachea. We are in the zone of the opposite recurrent laryngeal nerve. We will take off this isthmus and a small portion of this right lobe. Early in my experience I was called suddenly one morning to see a girl who was choking to death. The location of this isthmus and its firmness made me think of that case. The girl was cyanotic and scarcely breathing. I found that she had a sudden enlargement of her thyroid.

She was unconscious. I had my pocket case with me and I cut into her trachea and divided the isthmus of her thyroid, which was constricting the trachea and choking her. After this she began to breathe regularly, and subsequently the goiter disappeared completely. That case is reported in the literature, and in the report I called attention to the fact that the goiter disappeared after division of the isthmus. We never were able to satisfy ourselves as to the reason for it, but the clinical fact remained that it did.

We will place a clamp on the right lobe. In this procedure there is practically no bleeding if one keeps on the line of cleavage. In operating on an exophthalmic goiter case we have no occasion to get into a bloody zone. It is very important to be on the true capsule in making this excision, and the work has to be done in a slow and careful manner in order to obtain good results. Here is another one of the parathyroids at the lower angle. We will now excise this portion of the gland, which we believe will be sufficient to give this patient relief from her symptoms.

We will make some sections of the gland to see if we can account for its sudden enlargement, and then we will have a careful microscopic examination made. The muscles which were cut will now be reunited with catgut. The wound will then be closed with deep catgut and superficial horsehair sutures, so as to secure accurate approximation, layer for layer. A small rubber tissue drain will be inserted and allowed to remain for forty-eight hours, or longer, if necessary.

In analyzing these thyroid cases you can make the subject of thyroid enlargement and thyroid management very simple. It is not at all complicated. If you go back to the embryologic or histologic elements of which the thyroid is made up for your starting-point, in all the lesions you will find that the thyroid is made up, first, of essential, specific, secreting epithelial cells which are arranged in tubules and acini on the cluster order. These columnar cells rest on a basement-membrane. Connective tissue forms the supporting framework of these acini. There is where you get the enormous percentage of lesions—in these cells and acini. Second, the thyroid also contains a large quantity of connective tissue which holds the gland together. If you could destroy all the connective tissue, the gland would look like a miniature cluster of grapes, the stems, however, being absent. The connective tissue binds the various parts of the gland together, and then there is, in addition to these two, the vascular supply, venous, arterial, and lymphatic, with no specific ducts leading from the gland, as, for instance, the chyle channels, to carry off the secretion. The secretion is taken up by the blood and not carried away by a duct. The gland, therefore, is made up of the epithelial elements in the acini of the gland, the connective-tissue framework, and vessels. Embryologically the epithelial elements are of what origin? The epithelial cells are of endodermic origin. The connective tissue and the vessels are both produced from the mesoderm. Malignant lesions of the thyroid are of two types only—one, the type that arises from the epithelial elements and is called carcinoma; the other, the type that arises from the connective-tissue elements and is called a sarcoma.

Let us go back to the most essential thing. Of the lesions that involve the connective tissue and the lymph-spaces between the connective tissue, you have what is known as metastatic infection in the gland, associated with grip, with typhoid, with scarlet fever, tonsillitis, with any of the severe infectious diseases. A suppurative tonsillitis may cause a metastatic infection in the thyroid, in which we believe the connective tissue plays an all-important rôle, and in which there is a hyperplasia of the connective tissue. In the acute stage the gland is enlarged and sensitive. There is elevation of temperature, and the patient is often delirious. The condition subsides slowly, and the swelling of the gland recedes. In the acute stage there are none of the intoxication symptoms that are associated with Graves' disease, the excessive production of thyroid secretion, or the excessive absorption of the thyroid secretion. When the inflammation in the gland subsides, exactly the same tissue changes occur as in the infections of any other loosely arranged glandular structure. There is cicatricial contraction of an excessive quantity of connective tissue, which, in turn, causes strangulation and compression of the specific thyroid cells. Such compression may result in complete destruction of their secreting power, so that after a patient convalesces from the acute metastatic infection of the thyroid she may develop typical myxedema.

The following is a case in point: The patient had tonsillitis. I treated her years ago for an enlarged thyroid. Later, after she married, she developed some mild symptoms of Graves' disease. Under arsenical treatment the condition entirely disappeared. About three years after this she was confined, and a year after confinement she had a severe sudden tonsillitis, with some soreness in the lower part of the neck. She had metastasis in her kidneys, and an intense toxic nephritis, with blood in the urine, for weeks, blindness, convulsions, and finally unconsciousness. She was relieved by blood-letting by her husband, who was a doctor. After he got her out of that spell she recovered. Her kidneys began to improve, and her edema, which was extreme in degree, because she was a stout, large woman, gradually commenced to subside. She went to a southern resort to spend the winter, and got down to 180 or 190 pounds in weight, when her edema began to get worse again. She began to gain in weight. This was in November. I saw her in the first week of the following March. At that time her urine contained a large quantity of albumin, but a relatively small number of casts. There was no blood in the urine. She had weekly or ten-day explosions, in which her abdomen became enormously distended, passed large quantities of gas, had a severe headache, with a temperature of 103° to 105° F., but more frequently 103° F. Gradually the temperature would come down to normal and stay there until she got another explosion. She looked like a large mass of blubber, filling three-quarters of the bed, and weighing about 300 pounds.

What was the matter with her? The microscopic findings in the urine did not justify the conclusion that all that edema was from her Bright's disease. Looking her over, I wondered what had become of the thyroid gland, that always had been enlarged. Examination revealed that the gland was not palpable. I concluded that it was probably a case of myxedema. Three-quarters of the hair on her eyebrows had disappeared; the hair in her axillary and suprapubic regions was gone. This confirmed the diagnosis of myxedema following contraction of the gland, the result of infection. We put her on sheep's thyroid and iodothyrin, and she fell in weight from 300 pounds to 120 pounds. She went to California the following winter, and developed an acute septic endocarditis, from which she died. She had always had a little albumin in the urine, but otherwise she was practically well. I cite that case merely to accentuate the contraction that takes place following infections of the connective tissue, and this means a destruction of the secreting cells of the gland. The nephritis complicated the case and made the differential diagnosis difficult.

I had another case that came from Nebraska during an acute attack. I saw the patient in a subsequent attack, in which the gland had contracted. She was fed on artificial thyroid.

In a third case of tumor of the tongue a remnant of the thyroglossal duct, which is embryologically the duct from which the thyroid gland is developed, and which opens on the surface of the tongue at the junction of the anterior four-fifths with the posterior one-fifth, the foramen cæcum. The moment I cut it open I recognized it at once as a colloid thyroid—an aberrant thyroid. I looked for another gland, but there was none. In the development of this thyroglossal duct the development of the thyroid proceeded downward to a limited degree and was arrested in the tongue end of the duct. The woman subsequently developed symptoms of hypothyroidism.

We began feeding her on sheep's thyroid, and kept it up for a year. Whatever remnant of the gland was left began to increase in size until finally she developed enough thyroid cells to carry her along without the thyroid we had been giving her. All symptoms of deficiency of thyroid secretion disappeared.

I cite that case that you may keep in mind the lesions which occur in connection with the connective tissue from cicatricial contraction, just as in the contractile form of cirrhosis of the liver. Connective tissue does the mischief.

Those are the principal lesions that occur in the connective-tissue elements. Now, as to the vascular lesions. There are lesions that occur in the blood-vessels, namely, dilatation and hyperplasia of all the vascular elements in the thyroid, and the result is a large pulsating thyroid without any symptoms of hyperthyroidism, without any symptoms of acute Graves' disease. There is no evidence of excessive production of the secreting specific, cuboid epithelial cells at all, but a great vascular pulsating mass, which bleeds severely when it is removed, but which is not associated with any of the dangers incident to removal of the Graves' type of gland.

The final and most important tissues in lesions of the thyroid are the acini and the cells that line the gland-lobules, that is, lesions of the epithelial cells and their basement-membrane. What are they? The normal product of the cuboid cells of the thyroid is what? Colloid material, and that colloid is produced and absorbed by a definite, automatic regulating system. When it is produced in excess of its absorption you have developed in the gland, if it is diffuse, large colloid masses, sometimes as large as a filbert, often larger, the typical colloid goiter, without any symptoms of Graves' disease. Then there may occur hyperplasia of the cells alone, with a perfect balance of absorption and secretion. When it is a circumscribed hyperplasia, you have an encapsulated adenoma—merely normal gland tissue. You often see this and can pick it out. Then you often have, in addition to that, excessive secretion within a distinct tubule, with encapsulation and retention of the secretion. That increases in amount and flattens the cuboid cells, making them squamous or flat cells. That changes the secretion from a colloid material to a serous material, and you have the development of serous cysts which often attain a large size. Again, in addition to this, there may be hyperplasia of the cells, a hyperplasia of the connective tissue, and the production and absorption of an excessive quantity of thyroid secretion, as we interpret it, with the symptoms of Graves' disease. We do not believe that it is a perverted thyroid secretion. We believe it is a normal thyroid secretion in excess of that used normally in metabolism, because you can produce the condition artificially, as in the treatment of adiposus dolorosa with thyroid extract, which is a specific for it. You can develop Graves' disease nowadays, all except the exophthalmos. You can get the tremor and tachycardia, and all that goes with it, by the feeding of an excessive quantity of thyroid. Is it a hyperthyroidism? Let the pathologist and the internist quarrel about that. For our purpose, excessive gland tissue and a sequential intoxication are produced.

There is, then, left only the other lesion of the epithelial cells, a malignant lesion—a carcinoma. All these conditions lead right back to the essential action of what? To the cuboid cells in their relation to secretion, to absorption, and to their multiplication as the result of some stimulus which we know nothing about. So you see, it is not at all complicated now. There are three sets of lesions. They occur singly, circumscribed, and multiple, depending upon how much gland is involved. In the management of these cases you have all these conditions to consider. If you have a vascular thyroid, with pulsation in the beginning, it is a case, except in the very young girl, that requires the removal of a large portion of the gland. In cases of ordinary hypertrophy of puberty, if you put the patient to bed, apply an ice-bag locally, and administer arsenic internally, the hypertrophy, in the enormous percentage of cases, disappears without any surgical treatment whatever, not only in one case, but in hundreds of cases, and many more patients than that, in our experience, have not needed an operation at all. If there is present in the gland a circumscribed cyst as large as an apple, a hen's egg, or an orange, and you put the patient to bed, apply an ice-bag to the neck, and give arsenic internally, no good will come of it. You might as well give anything else internally or externally—it would have the same effect. Our line of treatment must be individualized. The personal equation and the differential diagnosis must be considered and carefully weighed before you decide on what treatment you will adopt in a given case.

In the exophthalmic type of goiter medical treatment is in nearly all cases a failure, except in those cases occurring in young girls, and very young girls at that. The condition, as a rule, is a progressive one. Medical treatment at best gives only a temporary improvement. These cases ought to go to the surgeon. Therefore, if you make a diagnosis of beginning exophthalmic goiter, when there are present tremor and nervousness and tachycardia which come early, all these preceding the enlargement of the gland and the exophthalmos, that is the time to remove the thyroid, because then it can be done practically without danger. You avoid a long period of invalidism, you avoid the fatal conditions that otherwise would develop later, and which inhibit or prohibit operation.

Occasionally a patient comes in with symptoms of exophthalmic goiter, such as the tachycardia and the exophthalmos, but no goiter. I have had three cases of that type. There was no goiter, but they were said to have Graves' disease. Where is the goiter? Where is the thyroid gland? In the chest. In every one of these cases I was able to demonstrate the gland in the chest by percussion, and subsequently by the skiagram and fluoroscope. This is the vascular type of goiter. It throws an additional shadow in the upper chest, which is apparent in the skiagram under ordinary circumstances. The gland is always there. Sometimes there are manifested pronounced symptoms of exophthalmic goiter with a very small gland. Bear that in mind. Remember, it is not the quantity of thyroid secretion that is absorbed, nor the gland that secretes the most thyroid, that causes the most severe symptoms. It is the lack or loss of balance between the requirement of the body for, and the production of, thyroid secretion. Just as soon as that balance is lost, exophthalmic goiter develops, regardless of the size of the thyroid.

In the management of a case like this you go back to the management of the acute infection. You prescribe an ice-bag locally and rest in bed. An acute infection of the vascular type, if excessive, must be treated surgically.

Then we come to the colloid type. Once the gland attains a considerable size and is fixed, the patients do not, except young girls, as a rule, do very well under medical treatment, and the removal of the gland is required. There are scattered throughout the gland masses of encapsulated colloid material as large as a filbert, which are not absorbed. Occasionally the gland enlarges synchronously from two conditions. First, growth and development of the epithelial elements in the acini, with the formation of new atypical acini, and, second, a malignant lesion may develop in another zone; then you have all the dangers that go with the malignant lesions of the thyroid.

The management of the hyperplasias of the thyroid in young girls is always medical. When the gland becomes firm and hard, the case is a surgical one and without danger to the life of the patient.

In the management of cysts, and particularly cysts of the isthmus, or one or the other lobes of the gland, enucleation of the cyst without removal of the thyroid is the operation to be done. There is usually present with it a thyroid of normal size, which is not removed. Simply enucleate the cyst with its capsule. That is all there is to it.

Then there are those large swellings of the gland that occur deep down behind the sternocleidomastoid muscle, and occasionally under the sternoclavicular articulation, which may suddenly endanger the life of the patient. I recall a case of that kind occurring in a doctor in this city. He had a cystic goiter; suddenly it began to distress him. He rushed to the West Side Hospital, and Dr. A. H. Ferguson immediately incised it, put in a tube, and saved the patient's life. He had a mild infection following that, but the wound closed up, and the doctor remained well.

When there occurs a sudden swelling of both lobes, strangulation may take place because of constriction of the trachea by the isthmus. You make division of the isthmus in such a case, lift the trachea and isthmus out from the substernal space, and the patient is relieved.

Now, as to the operations for the removal of the gland in exophthalmic goiter. Here one must individualize in a most careful manner before deciding whether or not to operate. These patients come to us in all stages of the disease, from the mild or comparatively mild type to the very severe type, when the patient dies the day after arrival in the hospital without having been touched. It is useless to send a case in this superlative degree of exophthalmic goiter to the surgeon. If it is a case in the superlative degree, keep the patient at home, in bed, flat on her back, put an ice-bag on the neck, and administer arsenic. Put the patient mentally and physically at rest, and if she can be saved at all, she can be saved in the comfort and mental ease of her own home. If you expose her to the dangers of transportation and the anxiety that she feels about having an operation, she will die shortly after she arrives at the hospital, before anything at all is done, or on the way thereto, as we have experienced, and as the Mayos have experienced very frequently. The best treatment for hyperthyroidism of the acute and virulent and dangerous type is absolute rest in bed, with an ice-bag on the thyroid. Nothing else compares in value to that. That has been well established by Kocher, by Crile, by Mayo, by Moynihan, and by all the men who have had large experience in these thyroid-gland lesions. When the patient comes out sufficiently from that acute condition, you can commence the milder operation of ligating the superior pole, which is always associated with more or less danger. Why? Just the mere fact that you are only going to ligate tells you why. Because the patient is in so bad a condition that you do not do anything else, and if you do that, you are likely to pay the price with a fatality. If you look at the statistics of ligating the superior pole of the thyroid, you will find that the mortality is greater from it than from removal of the gland. In a case in which we operated a few days ago, we ligated the superior pole under local anesthesia. We did nothing else. That night the patient's temperature was 103° F. and the pulse 148. Why? Because he was very sick before we operated—he was intoxicated with his hyperthyroidism, notwithstanding the fact that he was for two months on the ice-bag treatment and rest in bed. He almost died at one time from a bowel fermentation of a mild degree, but in a week he came back again to the condition he was in before. He now has an occlusion of some of his thyroid vessels, and a lessened secretion from his thyroid. Later we will ligate the other pole and secure sufficient diminution in the arterial pressure to get some connective-tissue development and contraction of the thyroid, and after that we will have to prepare for the removal of the other half, if necessary. The ligation of both superior poles, if the patient survives the operation, is not without danger.

I operated on a patient in February. Following the ligation, the patient made a nice recovery, notwithstanding that her's was an extreme case. The gland commenced to get hard, and we let her go home. When I returned from a European trip in August her feet and hands had begun to swell. She had albumin in the urine. She was brought back to Chicago. The hair had disappeared from the axillæ and the symphysis pubis, and as a result of the ligation she had progressive thrombosis in her yet enormously large thyroid. It was still a large thyroid, but it was as hard as marble from the thrombosis. All the secreting cells in the gland had been obliterated by the thrombotic process,—just what we wished to secure in a minor degree,—but this condition reached the superlative degree, and before we could place her under artificial feeding of thyroid she died, showing that when you ligate successfully you are still not free from the danger of the thrombophlebitis destroying all the gland-cells. If one is on the alert, however, and begins artificial feeding early, he saves these patients, because the thyroid glands of children, dogs, cats, and all sorts of animals can be removed and the subsequent result controlled absolutely by thyroid feeding.

In the operations for exophthalmic goiter one must be cautious as to the time of operation. He must individualize his cases. If the heart muscle is so affected that the pulse ranges between 160 and 180 at the time of the operation, or for days preceding it, postpone the operation until this condition is improved. It does not matter to the surgeon whether the heart-cells or the Purkinje cells (Crile) are mostly damaged, clinically the fact remains that these patients do not withstand surgical attacks well, and they must not be operated on until they are prepared and ready for operation. We did not have to take that matter into consideration in this case at all. This patient had compression of her trachea. You saw me pulling up the gland from the side of the trachea, where it was caught under her sternoclavicular junction and compressed the trachea, being followed by an acute edema or hemorrhage into the gland, and that developed the condition she had. We did not have to fear her heart muscle or her Purkinje cells. We are not afraid that when we go upstairs she will die if we talk to her, as some of these patients with acute Graves' disease do after operation.

With reference to metastases of this gland, when these patients have malignant neoplasms they metastasize in different places. They metastasize in the bone. If it is a sarcoma, the metastasis occurs in the long bones. If it is a carcinoma, it also occurs in the long bones. If it is a metastatic normal thyroid reproduction which occasionally occurs, one which is not malignant, it always occurs in the flat bones, as in the calvarium. If it appears on the skull, it is a clean-cut thyroid metastasis of normal histologic structure—it is not malignant.

The treatment of these conditions is obvious, and we will not discuss it at this time.

DOUBLE CERVICAL RIB

HISTORY

Patient is a female, aged thirty-nine, a teacher by profession, who enters the hospital on account of a dull, aching pain in head, which radiates down the right arm.

Family history negative. Has always been well with the exception of ordinary diseases of childhood.

Present Trouble.—Commenced in April, 1911. The patient states that the right half of the head became numb. This numbness was accompanied by entire loss of sensation. This condition slowly improved and entirely disappeared in two weeks; then the shoulderblade on the right side became painful at intervals, and there was a spot near the inferior angle of the scapula, 3 inches in diameter, which felt cold. At this time the arm became numb and she could barely feel any sensation when any one touched the hand or arm. This was particularly noticeable on the thumb and first finger. The numbness gradually disappeared, but the arm was weak, became easily fatigued, and was cold most of the time, especially during the night. The ache continued in the shoulder during this time. In August the arm lost its feeling of numbness. This was four months after the onset of the trouble; the circulation became better, and the pain in the shoulder disappeared. The thumb and the first finger are still numb. The sensation in the rest of the arm is normal. The arm and hand are inclined to feel hot at the present time.

The other arm has given no symptoms of pressure. There has been no swelling of the arm at any time.

COMMENTS AND OPERATION

This patient complained of pain in her right arm, and particularly in her index-finger and thumb, in the zone supplied by the musculospiral and median nerves. She has consulted a number of physicians for the trouble, and they all informed her that it was a neuralgia or a neuritis, and without endeavoring to ascertain the etiologic factor in the neuritis, it was prescribed for. A careful examination of her supraclavicular space shows she has a prominence above the first rib, and that this prominence extends forward to the posterior margin of the scalenus medius muscle; over this prominence on each side can be felt the pulsating subclavian artery. The least compression of this artery suppresses the radial pulse. Pressure of any severity causes pain to extend down the nerve-trunks, and particularly to the thumb side of the hand and forearm. There have been no trophic changes in the muscles supplied by the musculospiral or the median. It is clearly demonstrable, even without the *x*-ray, that the neuritis in this case is due to the pressure of a right seventh cervical rib; that the pressure has not so far interfered with the left brachial plexus or artery.

In the clinical history of cervical ribs we have two distinct sets of symptoms, depending upon the structure which is most interfered with by the advancing end of the cervical rib. The first set of symptoms is referable to the compression of the subclavian artery against the scalenus anticus muscle by the end of the rib, and produces ischemia of the arm and forearm, particularly at night or when the patient is compelled to hold the arms in the tense position for any considerable length of time in the daily occupation. The second train of symptoms is due to compression or irritation of the trunks of the brachial plexus. These trunks are also pressed forward against the scalenus anticus muscle, which causes the neuritis or neuralgia of which most of these patients complain before they have the ischemic symptoms. While the condition is congenital, it rarely manifests itself in the way of irritation until after the twentieth year. It is most marked between the thirtieth and fortieth years. The ischemia can be so pronounced as to cause gangrene of the fingers and thumb, more commonly of the latter. It rarely ever produces a complete paralysis, but any of the group of muscles supplied by the brachial plexus may be affected.

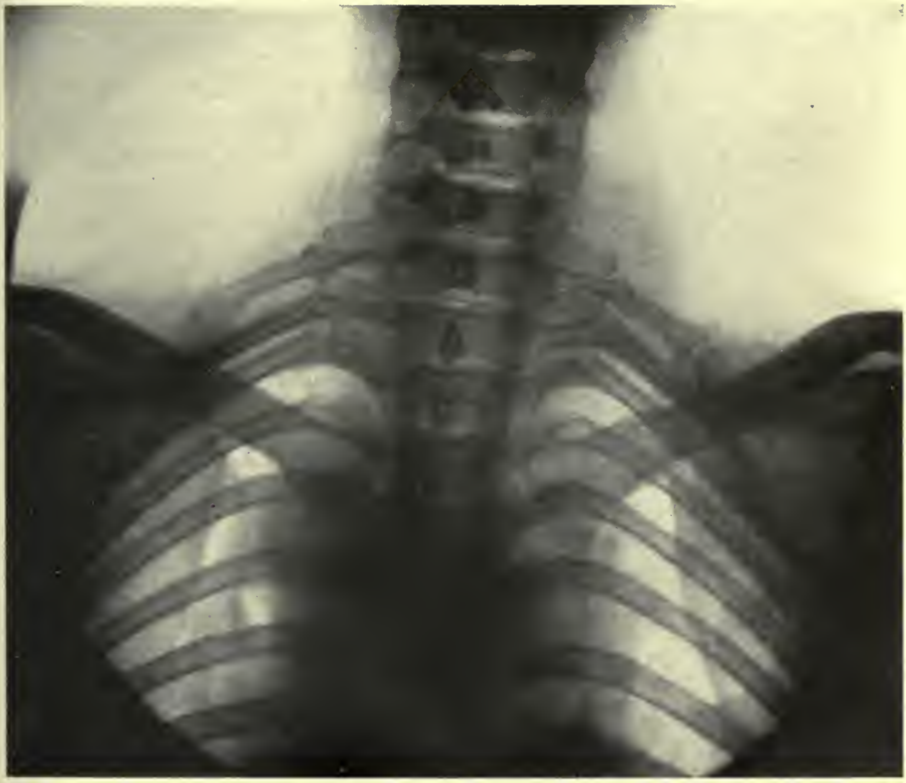


Fig. 74.—Skiagram showing double cervical rib.

The treatment of this lesion consists in the removal of the supernumerary seventh cervical rib. This should never be attacked from the apex, as on its apex are situated the nerve-trunks and the subclavian artery. The subclavian vein is never compressed by the supernumerary cervical rib, because the subclavian vein is situated anterior to the scalenus anticus muscle and cannot be caught and compressed by the advancing rib; it is displaced forward, but never to the degree of producing stasis in the vein. For that reason these cases are never associated with edema of the arm or forearm, and this accounts for failure of diagnosis of the disease in so many cases when really it should accentuate its probability. It can easily be attacked from its base by making an incision over and parallel with the anterior border of the scalenus medius muscle; one comes readily onto the neck of the seventh cervical rib; it is freed outward toward its tip, retracting the vessels and nerve-trunks inward. The neck of the rib is then divided with a short bone-cutting forceps, and the shaft grasped with a sequestrum forceps and the rib avulsed. The cartilaginous tip which remains attached to the first rib after the avulsion can easily be snipped off with a rat-toothed rongeur forceps. A deep muscle suture is made, and the skin is closed with horsehair. No drain. Subiodid of bismuth powder and a collodion dressing are then applied. The stitches will be removed in about ten days.

IMPACTED FRACTURE OF THE HEAD OF THE TIBIA WITH POSTERIOR LUXATION

HISTORY

School-girl, aged eighteen. Entered hospital February 14, 1912, because of old fracture of tibia and fibula with deformity. Family history negative. No past illnesses.

Present Trouble.—October 1, 1911, an express-train struck the automobile in which this girl was riding, killing two of its occupants outright, and throwing patient many feet. She was rendered unconscious. When next she remembered anything she was in bed. She was black and blue and sore from head to foot. The lower jaw was fractured, and the left side of head and face injured severely, but she apparently recovered completely from these injuries.

The right tibia and fibula suffered a crushing fracture without being compounded. She was in bed with a cast on this knee for seven weeks, and after that walked on crutches until Christmas, 1911. Since that time she has been able to walk, though with a limp, without the use of crutches.

COMMENTS AND OPERATION

Here is what has occurred: There was an impacted fracture of the external tuberosity of the tibia that split obliquely in a direction which permitted the occurrence of a luxation backward of the internal tuberosity, so that its anterior edge or margin of the internal condyle rests at the posterior margin of the external condyle of the femur. All the condyloid cavity of the internal tuberosity of the tibia is exposed behind the internal condyle of the popliteal space. Therefore there is an impacted fracture of the external tuberosity, with a rotation of the leg inward, so that the toe of the right leg, when standing erect, points toward the arch of the left foot unless a voluntary effort is made to rotate it outward.

She has good motion in the knee, but when you flex leg at the knee beyond 80 degrees, the posterior edge of the internal tuberosity impinges against the femur. When she stands up, she toes in. If she makes an effort, she can throw her toes out, but in the normal position she toes in, because of the rotation inward. The accident occurred in October, 1911. Callus has formed. She has a fairly good joint. There is internal rotation of the leg, which is the most important impairment of its usefulness next to the backward luxation of the tibia.

The question arises, What are we going to do? We cannot bring these two tuberosities closely together because callus has filled that space. She has a movable joint. She has a joint that may be flexed almost to a right angle. We can divide the tibia below the line of fracture. If necessary, we can refracture the fibula and rotate that clear around into position without hazarding her joint one iota. We do not expose the joint by going into it. If we had this case originally, before callus had formed, we could have luxated the tibia back into position and put a nail clear across the surface, and thus have held it in position. We cannot do that now. The fracture has already united. We will take off the projection that is behind at the internal tuberosity, and which is limiting flexion, at a later operation, so that she will be able to flex her leg as well as she ever did. Remember, ideal surgery is that which produces results without hazard, locally or constitutionally. This is too good a joint to hazard now.

These skiagrams have great value in showing how important it is to make more than one skiagram, and how important it is to make the best skiagram possible. A skiagram is a part of the diagnosis now. It is a very essential part of the diagnosis. It is now a scientific proposition when you are doing this kind of work. Let me show you how important it is. There is here great subluxation of the tibia backward. The internal condyle is resting entirely external to the internal tuberosity. There is, you see, a very good joint. When she had the first picture taken

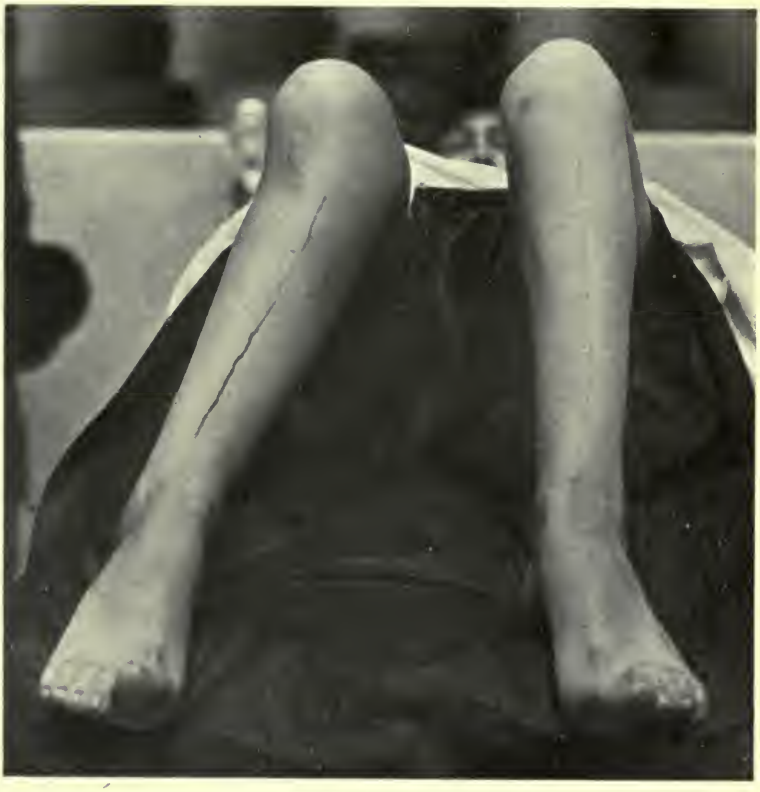


Fig. 75.—Before operation, showing deformity.

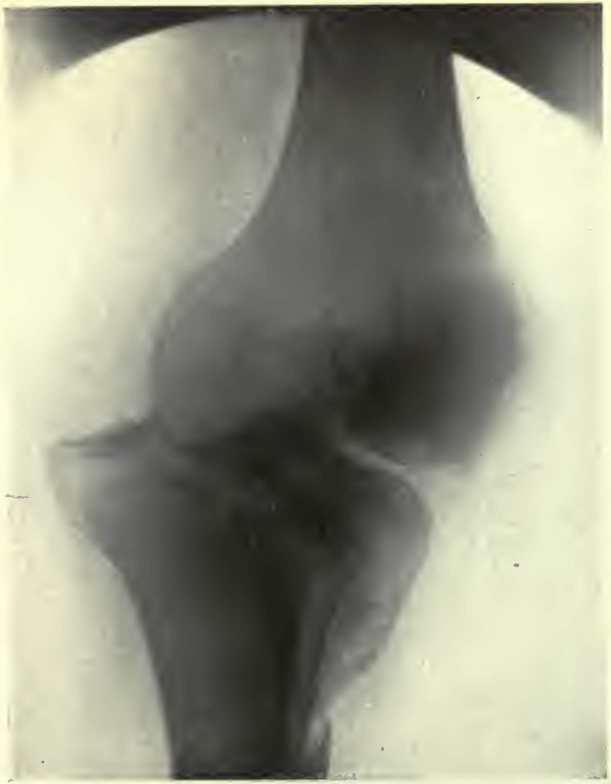


Fig. 76.—Skiagram of same case at different angle, showing lateral and posterior displacement.

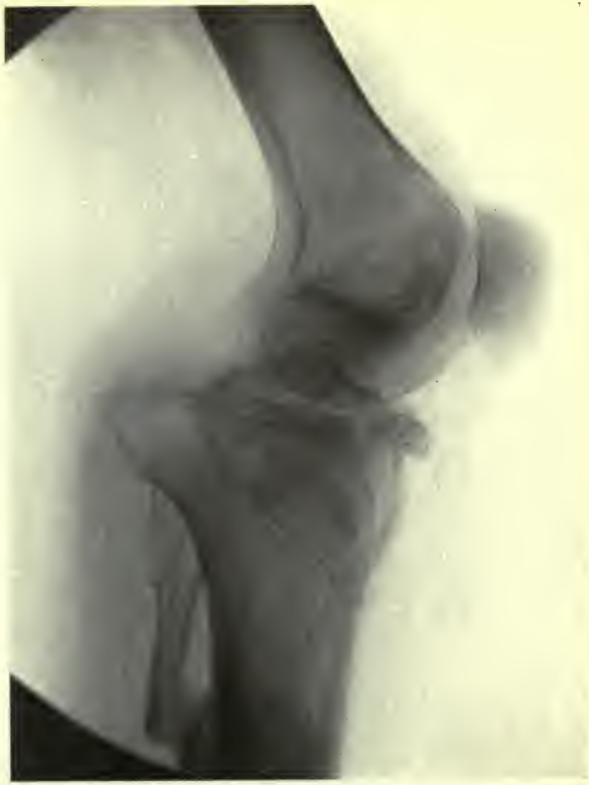


Fig. 77.—Skiagram showing fracture of head of tibia, with impaction and posterior dislocation.

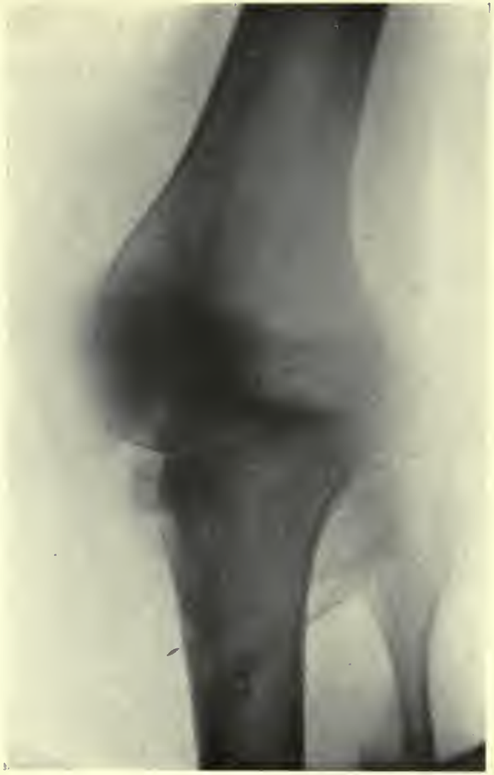


Fig. 78.—Skiagram showing apparent good position of bones.

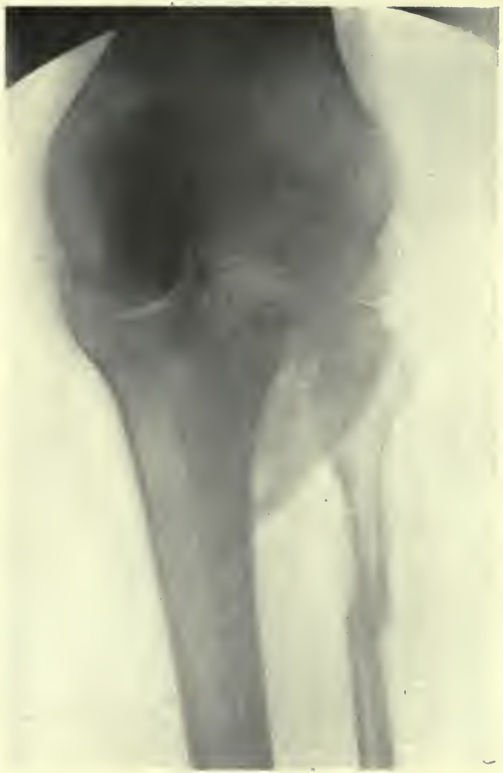


Fig. 79.—Skiagram showing apparent good position of bones.

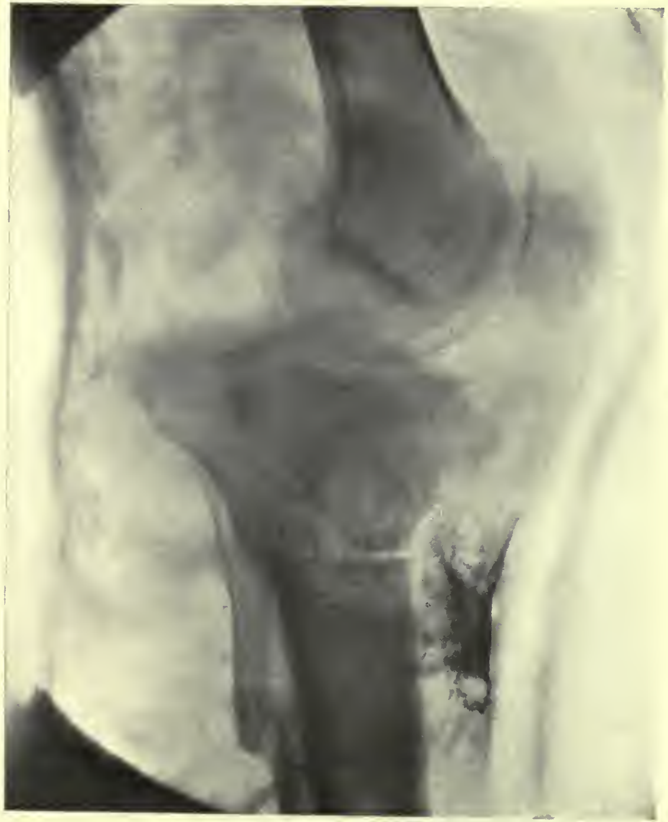


Fig. 80.—Skiagram taken through the cast four weeks after operation, showing magnesium splint in position. No attempt was made to correct posterior displacement.

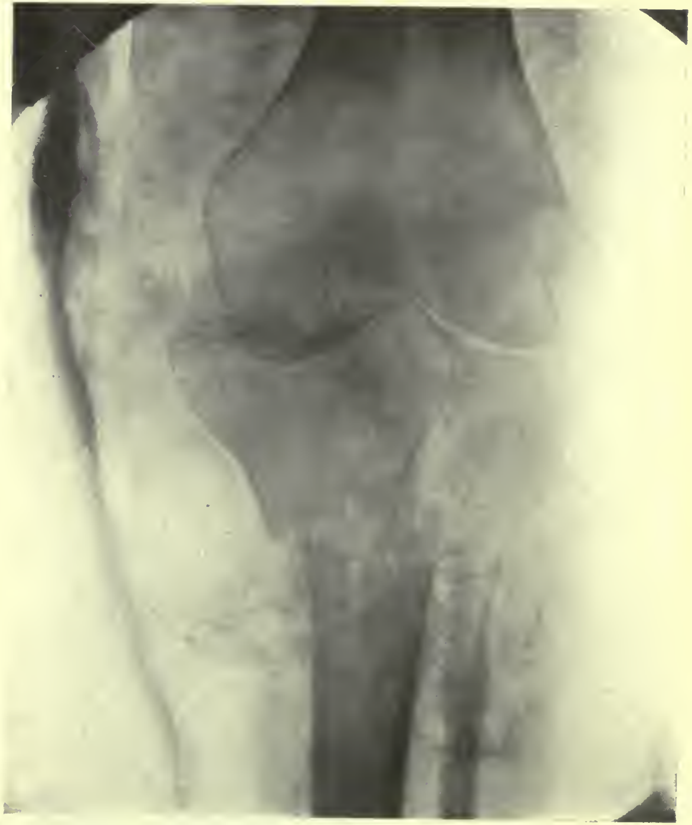


Fig. 81.—Skiagram taken through the cast four weeks after operation, showing perfect alinement.

one was led to say, "Why do you want to meddle with that bone?" But when you look at the two skiagrams made recently and synchronously, you can readily see how important it is to have these pictures taken in a scientific manner in order to get a proper estimate of the disturbance of relations. Let me show you where the greatest defect is. When you look at this joint from the side, you will see that the tibia is dislocated backward. You can bend the knee to a little more than a right angle. When you put the foot flat on the table, you see the foot is rotated inward and the leg is abducted, because it is shortened through the impaction on the outer side.

In this case we will have difficulty when we divide the tibia on account of the fibula, and on account of the external and backward luxation present.

There are some things about joints that we cannot yet control, and one of them is the fibrous type of arthritis following trauma, because it produces, first, a fibrous ankylosis and then a bony ankylosis. The fibrous type of arthritis is most common in the elbow, shoulder, and knee.

The tip of the tibial tubercle in this case is rotated inward, one inch from its normal location. What we want to do is to fracture the head of the tibia with chisel, make external rotation, and at the same time make abduction of the leg, and hold it in that position by means of a magnesium splint until we get bony union. We are going to fracture the tibia at about the place where it was fractured before—not in the same line, but on the same level, at its outer angle, because the same line would run up into the joint, and I am not going into the joint. The tubercle, the crest showing the line of division, the line of fracture, and here, as I elevate the periosteum, the line of luxation can plainly be seen.

I wish to cut across on about that level and make my division clear through the tibia, and rotate and abduct the leg. I will first cut the soft parts. This is the upper inner portion of the tibialis anticus. I am keeping far above the external popliteal nerve, so as not to interfere with its function at all. This is my landmark. You see, I have fractured the tibia just as I expected to do, exactly in the line and in the way I predicted. We rotate the foot outward. See my landmark! I know just how much I have rotated the foot. I have the necessary adduction. Let me recapitulate: The line of fracture was exposed and a transverse division made of the tibia through to the line; after the fracture the limb was forcibly tipped inward to produce an elongation of the fibular attachment. Rotation was then made of the tibia of just one inch—that is, external rotation of the lower portion of the tibia. When this was accomplished, the rotation was maintained by two magnesium interosseous splints, driven into the bone at acute angles to each other. These interosseous magnesium splints not only prevent a recurrence of the rotation, but aid in preventing the abduction of the limb; adduction will be controlled entirely by the plaster cast. When this union is complete, we will take off the posterior protruding internal

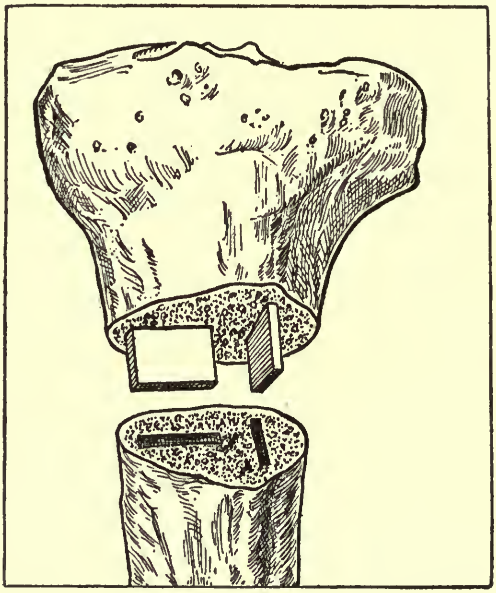


Fig. 81*a*.—Method of using magnesium ribbon splints in bones. Two splints in position in upper fragment with slots in lower fragment, into which splints will fit when proper approximation is made, thus maintaining separation and desired elongation as well as normal position.

tuberosity of the tibia. There will be an excessive bowing backward of the leg in that position, but that excessive bowing does not matter so long as the line is plumb in her leg. That is the most important factor. You can see that the joint has not been hazarded at all. We are not in the joint, and the joint has not been disturbed. I am going to overcorrect the leg in the direction of adduction, because it will come back some on account of tension on the ligaments. Deep sutures of catgut and superficial horsehair sutures close the wound. No drainage. Subiodid of bismuth powder is applied freely; the line of incision is covered with gauze wet with collodion, and then a plaster-of-Paris cast is put on, extending from high up on the thigh down below the ankle. This cast will be worn for three or four weeks, and then a long sole-leather leg and thigh case, laced in front, will be worn for several months. The patient will not be allowed to put any weight on her leg for at least six or eight weeks. Passive motion will be instituted after two weeks.

I put the bandage on this leg at an acute angle, so as not to get any too great tension if the knee swells. That bandage can stretch without becoming tight. You should never put on a circular cast without immediately cutting it, no matter what the acute condition may be. If you do not cut it, you will pay the price by having atrophy of the muscles or a myositis and atrophy. You cannot foretell when it will come, but it will surely come. It is only a question of time. If the cast is cut, it is perfectly safe. The magnesium is always absorbed, but as it is absorbed hydrogen gas is liberated, and this must be removed by aspiration. A piece of magnesium ribbon one inch square and about one-eighth of an inch thick will liberate about 15 gallons of hydrogen gas. The absorption of the gas is not productive of any bad results except the inconvenience it may cause through tension. It may be removed quickly by aspiration.

Outcome of Case.—Complete primary union occurred. The magnesium splint was decomposed and gas formed, so that at the end of three weeks it was quite puffy. The gas was removed with an aspirating needle. At the end of eight weeks the patient was permitted to put some weight on the leg. The limb was perfectly straight and the abduction of the foot was fully corrected. The skiagram shows the osteoporosis that occurred around the magnesium splint and also shows what appears to be a large granulation cavity. This is deceptive. It is the condition always present in fractures of cancellated bone tissue following union in the early stages.

TUMOR (HYPERNEPHROMA) OF THE KIDNEY

HISTORY

HOUSEWIFE, aged sixty. Entered hospital February 12, 1912, with hematuria as the presenting symptom. Family history negative as to malignancy and tuberculosis. She never has been associated with a tuberculous person, nor has she had any past illnesses. Menstrual history is normal. Menopause occurred at age of fifty, and she has not menstruated since. She has six living, healthy children.

Present Trouble.—In September, 1911,—five months ago,—patient began to have pain at times in the right lumbar and iliac regions. It bore no relation to the menses, which had stopped long before. It was sharp and aching in nature, and radiated down the thighs.

The urinations were accompanied by pain, and the amount has become increased, patient going to the urinal several times each night. In September, 1911, a small amount of blood was voided with urine for the first time. The amount was less than half a glassful. This, together with the pain, caused her to go to the doctor.

In November, 1911, in the middle of the night, she had a very severe attack of pain and vomited several times. In January, 1911, about the first of the month, she passed enough blood per urethra to fill a quart milk-bottle. Since then she has been so weak that she has stayed in bed continuously.

Her right iliac region, and just above and external to it, is the seat of great tenderness and pain. A tumor mass is present in this location. She had a chill one week before entering hospital, and cannot say as to the fever. Temperature normal on entering hospital. Examination of chest revealed a well-marked pigeon-breast. Heart was free from pathologic findings.

Cystoscopic examination February 13, 1912: "Very little congestion found about opening of right ureter, but a blood-clot could be seen escaping from it. No erosion or congestion of left ureteral opening. No sufficient evidence at right ureteral opening to justify a diagnosis of suppuration of kidney." Ureters were then catheterized, and 20 c.c. of urine obtained from each one. Urine from right side was much more cloudy. Laboratory findings as follows: Left kidney—numerous red cells present, 30 to 1/6 field; leukocytes, 6 or 7 per 1/6 field; a few granular casts present. Right kidney—erythrocytes, 12 to 16 per 1/6 field; leukocytes, 4 to 6 per 1/6 field. No casts found. Cultures showed presence of Staphylococcus albus. After repeated examination the tubercle bacillus was not found.

COMMENTS AND OPERATION

This woman first had blood appearing in the urine, and then she had severe colicky pains. She had nausea and vomiting, and the next day she passed, as she said, a sufficient quantity of blood to fill a quart vessel. You can see why she had nausea and vomiting. She had hemorrhage into the pelvis of the kidney, and had blood coming down through the ureter and distending it, and she had a reflex nausea and vomiting that comes with that condition. She has no history of having had chills; no history of fever; no history of having pus in the urine preceding these attacks of hemorrhage. Is that correct?

The Interne: Yes, sir.

Dr. Murphy (resuming): You have here the history of these hemorrhages first as the principal symptom, and pain as a secondary symptom only. This is in a line of work that Dr. Lewis, of St. Louis, who is with us today, and whom you all know, is particularly interested, and who has done such splendid work that I would like to ask him to talk to you for a short time on the diagnosis of this class of lesions while I go ahead with the work. I have great pleasure in presenting to you Dr. Lewis, of St. Louis.

Dr. Bransford Lewis, St. Louis, Mo.: Gentlemen: I feel that I am more or less trespassing to take up the time which might be so much better occupied by Professor Murphy. However, I am willing to do what I can in the line of duty.

The diagnosis of genito-urinary affections is somewhat different from the diagnosis of a great many surgical conditions, in that the accessibility of the urinary organs to the general practitioner is less than is the accessibility of troubles with joints and extremities, the face, and so on. But a short step to the reaching of these genito-urinary organs has been made within the later periods of development of surgery by various means of mechanical diagnoses, such as the *x*-ray, the cystoscope, the sound, and various other instruments. All these things have really advanced genito-urinary surgery and put it on a tangible basis, to my mind. A man with the analytic mind of Dr. Murphy can analyze symptoms and do it much better than you and I, so that the mode of reaching a diagnosis is not so easily attainable for us as it is for him. We have to make use of materialistic means for reaching a diagnosis, and these means are the ones I have spoken of. A diagnosis means more than saying whether a kidney is affected or not; we want to know *how* it is affected. For instance, let us take the diagnosis of prostatic obstruction. To say that a person has prostatic obstruction is a very immaterial diagnosis. We want to know a number of things about prostatic obstruction: we want to know whether there is simply hypertrophy or contraction, or whether there is a stricture or a cyst formation. The exact form of the obstruction must be known, as well as the location of the obstruction and the condition of the allied organs, the kidney, and so forth. The same way with gonococcal infection. We must know the exact location of the infection, and each and every location must be differentiated. These things are all illustrated, not, however, in an analysis of the history so well as through the exact physical examination, and determining where these infections come from and the parts that are infected, and how extensive is the infection.

With reference to the history of these cases, I have often thought how unfortunate it is that these patients do not have pain in the early period of their infection. Just before coming here I saw a patient who had hematuric attacks which were of the recurrent type for the last seven years, and this patient never had pain until the last month. The conclusion he reached was that it was necessary to consult a doctor and have his case analyzed, but he did not think it necessary to have this done until he got pain. Then, after seven years of trouble, he got pain and was willing to have himself investigated. This is an unfortunate feature in connection with these cases. They go through utter destruction of the kidney without having pain. I have seen the kidney with calculi in it absolutely destroyed without the patient ever having had pain—the kidney being absolutely destroyed with the absence of the symptom of pain. We must not, then, wait for the history of development of pain before we make a diagnosis in these cases.

I had one case recently in which the kidney was absolutely riddled with stones in a young man who was only twenty-six years of age. He had gone on for twenty years with bloody urine and a generally lowered condition of health. He never suffered very much from pain in his back. There were seven large stones in his kidney, occupying the kidney tissue from top to bottom, and without a history of pain. He had numerous diagnoses made, namely, general debility, gonococcic infection, and possibly tuberculosis, all of which diagnoses were incorrect, in that they were not based on the exact findings of the physical examination. In the examination of such cases one should demonstrate whether or not there is cloudy urine, which will lead one to examine the urinary organs from top to bottom. Examination with the cystoscope will show the presence or absence of trouble in the bladder; the ureters should be catheterized, and a determination made by means of the catheters as to whether the ureters are obstructed at any point, or whether there is a clear passage up to the kidney pelvis, drawing off the urine from each of the two kidneys, analyzing the respective urines from the kidneys, determining the rapidity of flow, as sometimes there may be one dram from one kidney and four drams from another. It is very essential to get an accurate analysis of the two urines from the kidneys.

Dr. Geraghty, of Baltimore, has lately developed an additional mode of determining the functional activity of the kidney by means of the phenolsulphophthalein test, which is accessible to all of us. By injecting 1 c.c. of this solution and having two catheters in the two kidneys, in five, ten, or fifteen minutes there is the appearance of red urine in the kidney. If one kidney is diseased, that will be delayed, whereas the healthy kidney will show it in five minutes. If you take two urines the first hour of excretion from the two kidneys, they give a certain percentage from the normal, namely, 40 per cent., and the second hour, 20 or 30 per cent. There may be a rather low percentage of excretion in the first and second hours after the administration of the drug. If it runs to 40 or 50 per cent.,—and this test is quite accurate,—there is definite physical evidence when you get pus urine from one kidney and healthy urine from the other. The pus may contain colon bacilli or gonococci. The use of these various modes of physical examination is now just as accessible to all those who make use of them as the use of any mode of physical examination for any trouble in the human body.

Dr. Murphy (resuming): You see, now, I have lifted this tumor of the kidney out. I have it in my field. You can see, here is the neoplastic growth at the lower end of the kidney. Here is the stump of the normal kidney tissue. Here is the pelvis of the kidney. This kidney is just about the size which makes it the limit of comfortable removal from this position. There is a cyst on the upper side. I am going to take this out to bring the pelvis of the kidney out into the field or forward. I have been able to lift up the upper pole of the kidney, as you see, and expose the dilated ureter, as you see it here. I will free that ureter from its neighboring structures, catch the vessels around it, and, remember, the ureter is always back of the vessel. In the embryology of the kidney you know that the ureter is back of the vessel. There is an artery pulsating in front of me. I have my finger on it now. Those who are near enough can see it pulsating. I know just where I am. I am freeing the ureter as it passes around the artery here. I will grasp that now and get it ready for a separate ligation. The ureter is not infected, so far as we know. This puts it on the distal proximal portion of the ureter. Now the ureter is divided. Here is the renal pelvis, and there are the renal vessels. We have all our field covered. I am trying this loose tissue which was attached to the kidney. We are down to the stump of this ureter. It has not been infected at all. It is perfectly dry. We have the whole thing in hand all the time. We tie the ureter. This tumor is either a hypernephroma or a sarcoma. It began in the medullary portion, and is almost confined to the medullary portion.

First, I will go through the technic of the operation, which I did not give, however, while Dr. Lewis was talking to you, and I take this occasion to express my appreciation to him for the discourse he gave you on the diagnosis and the significance of diagnosis in connection with tumors of the kidney.

The history of this case, you will notice, came along to the stage of hemorrhage. The stage of hemorrhage can occur in three lesions that are significant without having much pain at any time, but they have pain in varying degrees of early manifestations, dependent upon which one it is.

If you will begin with the stone type, these patients can have hemorrhage as an initial announcement of its presence without ever having had pain at all. But with the stone type the patient is much more likely to have pain and discomfort than with the two following types, which produce hemorrhage, and for that reason you expect it. But the stone may be large and impacted in the pelvis and never get to the stage of impaction in the ureter and give a typical colic.

The next type is tuberculosis. Tuberculosis can begin in the kidney, and the patient have only increased frequency of hemorrhage as a symptom of its presence.

The next type of tumor which produces hemorrhage without manifestation is the malignant type of neoplasm, and this is of the sarcomatous type, and in that we include the hypernephromata. These will go along for months and months without giving the symptom of pain, and finally the first indication of trouble you have is hemorrhage.

I want to show you this nerve. Look here! There is the nerve as it belongs there. As I pick it up I excite contraction of the muscle. If you include the nerve in the suture, you cause the patient a great deal of pain. This is the most common cause of pain after suturing in the removal of the kidney or doing a nephrotomy. Do not cut it. If you cut it you will leave an area of anesthesia on the upper portion of the thigh. If you include it in the suture, you will give the patient severe pain a few weeks after its inclusion, so be careful that you see it in every case. I see it with each succeeding stitch, and I am avoiding it all the time. It is situated at the lower part of the muscle here, so that there is no occasion for including it in the suture. I am approximating the muscles; I am closing around the vein with double suture so as not to compress it. It is a continuous stitch, and put in so as to get muscular apposition—and good muscular apposition should be carefully attended to just as one cares for the opening in the anterior portion of the abdomen. Remember that a hernia can occur here; that when the kidney is permitted to remain, you can have a hernia of the kidney there and a very annoying condition.

In doing the operation we first cut down anterior to the quadriceps lumborum, open the fascia as it comes over, expose the cortex of the kidney, and free the kidney anteriorly and posteriorly above and below. In this class of cases you retain the capsule of the kidney. Even in a case of old infection you will find it of great advantage to leave the capsule in the abdomen and to enucleate the kidney from its capsule. In that way you come down onto the pelvis of the kidney with very little trouble. Where you have a suppuration in a large cystic kidney you not infrequently have adhesions high up in the course of the diaphragm, and you have great difficulty in getting them free. By taking it out in the manner in which I have described you have very little difficulty in freeing it, but if you take it out with the capsule, you have the greatest difficulty, and you may have a profuse hemorrhage in freeing the adhesions above the suprarenal capsule. When you come to the pelvis of the kidney: when you have removed the true capsule, you must not endeavor to ligate the vessels while the kidney is in position. You must use a rectangular forceps, such as I am using here, and this has been in use for many years. It was not used when I was an interne. After you have taken the kidney out without its capsule, having enucleated it within its capsule, you cut down to the pelvis and loosen its capsule outward from the pelvis of the kidney, and that leaves the vessels. You can put forceps in here, anteriorly and posteriorly to the pedicle, and you slip this onto the pedicle and locate it from below. You do the same from above, and then you cut off the kidney and can have the vessels exposed. You can go on and ligate the arteries, if you wish, or do as I have done so often, disconnect the handles of these special forceps and extract them and let this stay on for a few days, and by that time thrombosis has occurred and you have no trouble with the ligatures. It is one of the easiest of subcortical enucleations. When you take out the true capsule of the kidney with it you have little or no trouble in exposing the vessels; you are not in danger of tearing the vessels in elevating the kidney. If you do a subcapsular enucleation you can tear off the vessel and have a fierce or profuse hemorrhage.

In doing one of these operations and removing the sac of a suppurative cystic kidney which had burrowed inward along the renal vein to the vena cava, I had to make a large incision, and I was peeling the sac off from the vena cava and tore it. In looking at it I simply put on three hemostats, went on with my enucleation upward from that, and I did an overstitch of the vena cava. You do not have to ligate any of the large veins. A simple suture stops bleeding from any large vein. Remember, the larger veins have less blood-pressure, and many of them have a minus pressure on inspiration and a slight plus pressure in the vessels of any portion of the trunk. It is a different proposition where you injure an artery. I went on with my enucleation, completed it, and the patient had no unpleasant symptoms.

There are some things about taking out a kidney that I wish to refer to. In the first place, in a large kidney like this, where you have a tumor in the kidney, you are not permitted to put on these forceps for delivery. If you do, you press the tumor and force the contents of it into the veins and disseminate it through the system and develop a fatal condition for the patient.

The way in which we elevate the kidney is to free it all around above and below. After we have freed the kidney all around,—let this represent the kidney [indicating],—I take and put that down in to the lower pole, pass it in front of the vessels and under the pole, and bring it around posteriorly. You see what I have done. I have a network. That is what I call my sling elevation. You get around and you can easily lift it out into the wound without the slightest danger of lacerating vessels. It is of the greatest assistance where you are going to do an operation on the pelvis of the kidney to throw a strip around and lift it out. You do not traumatize the kidney at all. You do not disseminate the contents of the neoplasm through the veins as you do if you produce traction with the hands or with an instrument in effecting delivery.

The next thing is to free the ureter. The pelvis of the kidney is behind. The artery is divided in two branches. This point was brought out by Israel, the upper branch going around the pelvis, and another going behind the lower half of the renal pelvis. When that condition occurs, as you expose the pelvis you see the renal artery in the field. You cut it and ligate it and go on with the enucleation of the pelvis, bearing in mind that the large vein and large artery are always in front of the pelvis. Therefore, when you wish to do any work on the pelvis of the kidney you attack it from behind, not from in front.

In making a diagnosis in this case we had hemorrhage without pain. We had no evidence of calculus; we had no evidence of tuberculosis, and we had no evidence of a discharge of pus irritating the neck of the bladder. That was the principal thing that might lead us to conclude quickly that this was a neoplasm. If she had a tuberculosis, or if she had a calculus in the pelvis of the kidney along with an irritating discharge from the pelvis, it would have eroded the zone around the ureter. There is no zone of erosion around it, but merely a blood-clot protruding from it.

As we take up the specimen and analyze it we see that the tumor began either in the pelvis or in the cortical portion of the kidney; it looks like a hypernephroma, and I rather think it is.

It began as you see, and gradually compressed the cortical portion of the kidney, so that there is less secreting surface than one would expect.

I will make another incision through the tumor and see the color of the remaining portion of it. It is a sarcoma or hypernephroma, and not a carcinoma. The first symptom she had was blood. Then came the attack of pain, with a large quantity of blood, but hemorrhage was the first symptom. I should say, from the gross appearance of this yellow mass, that it is a hypernephroma. The saving of this kidney was out of question.

What is the immediate prognosis, so far as the danger to life is concerned. It would be about 5 per cent. of danger, because there is a considerable portion of the kidney that remains which is normal kidney tissue, and the danger in removing a kidney, as far as the suppression of urine is concerned, is in direct ratio to the degree of normal structure still remaining. If the patient is perfectly well and has a kidney removed, when the cortical substance has not been destroyed, as is occasionally done with division of the ureter, the danger is approximately 30 per cent. There is suppression of urine, throwing all the labor onto the other kidney. Every one believes now that I am placing that estimate too high. I am positive I am not, that is, where you have a practically normal kidney. Where you have a kidney that is destroyed to a shell, with a big abscess as large as a slop-bucket, the danger of that is about 2 per cent. Just think of the contrast! The more kidney there is destroyed by the disease, that is, the secreting substance of the kidney, the more favorable the removal of the kidney is, because the work is actually thrown onto the other kidney and the other kidney readily assumes it.

You doubtless remember the splendid series of experiments which were made with reference to excessive secretion from resection of the kidney. It has been clearly demonstrated that if you take out two-thirds of a dog's kidney or a cat's kidney, and finally all of it, the other kidney, within a limited period of time, approximates almost the exact weight of the combined kidneys. It takes on the labor without a particle of trouble if it is thrown onto it gradually, but where it is thrown on suddenly, then the patient has edema and swelling of the kidney under a normal tense capsule, and that kidney stops secreting by a compression strangulation, the patient has suppression of urine, uremia, and a fatal termination. Occasionally you may tide a patient over by doing phlebotomy and extracting a large quantity of blood, which lowers the arterial tension, lessens the tension on the cells within the capsule, and gives the secretion an opportunity to escape.

If there is any place where capsulotomy would be indicated, it is in that class of cases, in the acute congestive lesions, or in the acute infarcts which completely occlude the artery on one side and stop secretion on one side. If you will divide the capsule and permit the kidney to secrete, you occasionally tide the patient over a few dangerous hours, when he or she, as the case may be, will have uremia and die.

The final prognosis in this case is entirely different from the immediate prognosis. The final prognosis, if it proves to be a sarcoma, is that the patient is eventually going to die from sarcoma in the other kidney. Many years ago we collected statistics of a large number of cases of sarcoma of the kidney; this was before the time we differentiated hypernephroma, and before the time we differentiated the small round-cell neoplasm which so closely resembles sarcoma, and that we had not, up to that time, classified. With these two exceptions every single case of removal of the kidney for sarcoma had, within a period of twenty-one months, gone on to a fatal termination or to the development of a perceptible tumor in the other kidney. I do not know what Dr. Lewis' experience has been, but my experience has corresponded with those facts. Wherever I have removed a sarcoma of the kidney there was always a return of the sarcoma in the other kidney. The hypernephromata occasionally give us a permanent cure, but not often. The hypernephromata metastasize not infrequently very early in the manifestation of the disease, and you have this metastasis in the lung, in the bone, and in the pleura before you have indications of its presence announcing itself sufficiently to call your attention to the lesion in the kidney.

I recall a very bitter lesson I had, after reading the article of a Scotchman, in a child that had been under my care. In the case of this child I made a diagnosis of sarcoma of the kidney; the tumor grew slowly. For a year I did not see the child, but in the mean time the tumor had grown a little larger, and at the end of two years it was still larger, and in three years I put a large interrogation point after my diagnosis. In four years the interrogation point became larger, and in five years I concluded I was wrong. I did not take out the kidney because I felt it would not prolong the patient's life to do so. However, concluding that I had made an error in diagnosis and that the neoplasm was some other type than that of a sarcoma, I took the tumor out, and within six weeks the patient had a tumor larger than the original one, and inside of twelve months she was dead. As long as I let it alone the patient was doing apparently well, but the moment I touched it, it immediately started out and produced its metastatic and local recurrence. It was an endothelioma, and endothelioma is the other type of tumor in connection with the hypernephromata. We do not know exactly where to put these endotheliomata as yet. They have no place in the classification of tumors. From a practical standpoint, however, you can put them with the sarcomata, particularly where the endothelioma occurs in the renal structure; the tendency is to recurrence of the same type of tumor in the other side.

So in this case, finally, the prognosis is very unfavorable.

CHOLELITHIASIS

HISTORY

PATIENT first visited doctor on February 21, 1912, on account of severe pain in the right hypochondrium, radiating to the back and up to right shoulderblade. He stated that about two years previously he was seized suddenly with a similar pain, since which time he has had frequent attacks of varying duration and severity. The recent attack began on February 18th, and has persisted ever since in spite of large doses of morphin. He has never had any chill or fever and never noticed any jaundice or change in the color of the stools or of the urine. On February 21st he was apparently suffering greatly, but the physical examination was entirely negative, except for tenderness in the right hypochondrium on very deep palpation. There was neither rigidity nor a palpable mass. The urine obtained at that time was acid, pale straw color, specific gravity, 1020, contained neither albumin, sugar, nor bile. Microscopic specimen negative in the centrifugalized specimen for pus, casts, or blood.

Doctor was called again to see him February 23d, on account of a severe attack of pain which had disturbed his night's rest. The examination this time revealed a temperature of 99° F. and a well-localized tenderness in the region of the gall-bladder. A specimen of stool was negative for blood both microscopically and chemically to the guaiac, as well as to Myers', test. There was an abundance of meat-fibers, but no excess of fat or fatty acid crystals. A test-breakfast has not been obtained. The diagnosis has rested between cholelithiasis and duodenal ulcer.

February 23, 1912, leukocyte count 22,000. Coagulation time, 2 minutes 18 seconds. Temperature on entering hospital, 99° F.

COMMENTS AND OPERATION

Why do we operate on this patient this morning in his acute attack? He is not icteric. He has no elevation of temperature, but he has a leukocytosis of 22,000. What does this mean? It means that he has an infection; that there is absorption of septic products; that he is putting up a good fight against it, and that he has some acute retention in his right hypochondrium. That is why we operate today. He came to the hospital last night with a history running over a long period of time. He has had retention in his gall-bladder for eight days. He is still having pains. If there is obstruction by a stone, it is in the cystic duct. The stone has not gone into the common duct. If you are going to save him from graver dangers, you should open the gall-bladder now, before the stone is forced into the common duct, and before he gets infection in the common duct with a stone, because it was such cases of stone in the common duct with infection that gave Kehr a mortality of 90 odd per cent. You do not have to operate in the acute attack where the stone is in the common duct and icterus is present. Where there is no icterus the stone is not acutely impacted in the common duct, because the patient has no bile in the urine and presents no evidence of icterus. The man has no elevation of temperature, and unless you made a leukocyte count you would have no evidence of infection. The leukocyte count tells the story. It is an important factor in aiding you to arrive at a diagnosis in this class of cases, and in the appendiceal cases where you have but little elevation of temperature, say a temperature of 99¾° or 100° F., but a leukocyte count of from 18,000 to 28,000 or 30,000. In this case it is of real value in the way of differentiation; it is not so important in the other class of cases. Let us figure this case out.

This man's brother came to me two years ago after many weeks of suffering, with a large abscess in the right hypochondrium, and with rupture of his gall-bladder from gall-stones. I opened the right hypochondrium and drained the abscess, and removed a number of stones which were extracystic. I did a secondary operation later, removing some stones which escaped from the gall-bladder at the time, and removed the gall-bladder some months after that. Why should I let this man go on to the superlative degree of pathology as his brother did if we can open, drain, and remove a calculus and thus obviate a pericholecystic abscess, which might develop subsequently?

In making a differentiation between lesions that occur in this position you have to consider, first, the gall-bladder; second, stones in the kidney; third, the appendix; fourth, acute infections of the head of the pancreas; fifth, acute ulcers of the duodenum and stomach. You can rule out acute ulcers of the duodenum and stomach because they rarely ever cause the severe type of pain you have in the gall-bladder infections. I recall one exceptional case, a policeman, who came in here a number of times reporting that he had had recurrent attacks of acute abdominal pain. Finally he came in on the first day of one attack, and one could see, from the location of the pain, that it could not be the gall-bladder that was at fault—it was the stomach. It was a stomach ulcer which would get well between the attacks, it being very much like the crises that occur in tabes, except that he was more sensitive to pressure in his epigastrium. Involvement of the urinary tract—a urinary calculus—is a condition you can easily differentiate by the fist percussion method which I have demonstrated here repeatedly. (See p. 459.) It is the best physical sign I know of in differentiating abdominal lesions, and it aids more frequently in making a differential diagnosis in the way of excluding or including the kidney in the lesions than any method I know of.

Choleliths result from an entirely different condition than that causing urinary calculi. A cholelith is the result always of infection in the gall-bladder. The bulk of material which goes to make up the cholelith is cholesterin. The cholesterin is not precipitated from the bile that has been carried into the gall-bladder, but it is the result of degeneration of the epithelial cells of the mucosa of the gall-bladder produced by the infection, and precipitated and crystallized in the gall-bladder.

I am making a long oblique incision, 3/4 inch below the costal cartilage margin, over the rectus muscle, and down the linea semilunaris, making the incision entirely through the sheath of the rectus muscle. The fibers are split and displaced and the peritoneum opened. Here is the gall-bladder exposed.

The gall-bladder is not ruptured. It does not look like a gall-bladder. It is pale pink, acutely infected and infiltrated, and looks like an inflamed stomach. This is a typical picture of a case of acute infection. Note the pale pink color instead of the normal slate color. It looks like a peach. I cannot even tell whether or not he has a stone, on account of the tension and non-compressibility of the gall-bladder, and I am going to put in a coffer-dam of gauze. I will take the tension off of his gall-bladder first by aspirating it. The first bile extracted is usually clear, as the pus is usually situated down in the bottom or in the cervix of the gall-bladder. We have now the tension off of the gall-bladder, so that we can palpate the neck of it. I find there is a large stone in the bottom of the gall-bladder. It has slipped by, and it feels as though it is impacted in the cervix, but it has not ulcerated through. With a little manipulation I am able to free it from this hour-glass pocket and press it back into the gall-bladder. We are getting ready to make closure of the gall-bladder before we permit this septic material to escape by placing two rows of catgut puckering stitches in its dome, ½ inch below the other. This stitch is going into the wall of the gall-bladder but not all the way through. I am going to make an entropion of the cut edge of the gall-bladder and drop it back into the free peritoneal cavity.

When I wrote my first article on gall-bladder surgery and the use of the button in that connection you could put all the recorded cases on a single page of ordinary paper. The button was originally intended for the operation of cholecystenterostomy, and was used primarily for that purpose. Now, of course, its use for that purpose is rather rare, since we have developed gall-bladder surgery. Kocher, in his latest work, says that while the button has disappeared to a certain extent from other fields of work, still, where you are unable to clear the duct and wish to do a cholecystenterostomy, the button holds a preëminent position. For posterior gastro-enterostomy we still use the oblong button because we believe we can do the operation better with the button than with other means.

We will put in a second row of sutures outside the first, because the inflamed gall-bladder will not yield like the ordinary soft gall-bladder does. We have protected the field of operation by sponges, and we are now going to open the gall-bladder and remove the cholelith. So infiltrated is the gall-bladder wall that the opening does not contract. Here is a very pretty example of necrosis of the mucosa from the continued ischemia and pressure of the retained product under tension. Now comes the débris from the retained infection product. Every now and then a considerable quantity of pus comes out. We broke this stone in displacing it. That is the one that got down in the cervix fellæ first. The smaller ones could not pass by it. If they could have gotten by, he would have had recurrent attacks of jaundice.

We inject atropin in most all operations preceding the anesthesia, particularly in adults, as it stops secretion in the throat and, we believe, lessens the liability to inhalation pneumonia, because it does diminish the secretion so markedly. One of the objections made to its use was that it did not help the patient to expectorate. They do not expectorate when they are asleep, and I believe that argument against its use is not a valid one. There is no question in the minds of those who have used it for a considerable length of time that it does lessen the quantity of secretion. We are notorious faddists in medicine, and we reserve the right to change our opinions. For the present that is my opinion, namely, that it does lessen the secretion.

We have the gall-bladder closed around the tube and the tube secured to the gall-bladder. We will put in another stitch, making it additionally secure, and we are through. We will commence to remove the packing which we had around the gall-bladder and remove with it the gall-stone débris and infected material that rest on the sponges. We want another tube for the outside. This is what we call the extracystic tube for drainage of the peritoneum, which we make use of in all the cases in which we have suppuration of the gall-bladder. This tube drains the hepaticoduodenal fossa. At the time we first described these operations we mentioned, first, the type of drainage and fixation to the anterior abdominal wall. Second, an ideal cholecystostomy, that of opening the gall-bladder, cleaning it out, closing it by a double row of sutures, and dropping it back into the peritoneal cavity. Third, opening the gall-bladder, draining it, securing a tube in it, and dropping it back into the peritoneal cavity. The latter process we denounced years ago, as we did not feel it was safe to drop the gall-bladder into the peritoneum in this position with a tube in it. Since that time the work of Mayo has emphasized that it is the safest plan, and we have adopted it. It is a plan that leads to very little trouble subsequent to the operation, as there is no fixation of the gall-bladder to the peritoneum. One of the conditions which we have, if we should fix the gall-bladder to the abdominal wall, as we did in our original operation, is the traction following the operation, which is painful and annoying and inconvenient to the patient, and we believe that is of sufficient moment to warrant one in doing the procedure which we are doing now, knowing, as we do, that adhesions form rapidly around the gall-bladder and rubber tube and that these adhesions protect the free peritoneal cavity.

The tube is left in three or four weeks, depending on the degree of infection. Here there will be considerable necrosis of the mucosa of the gall-bladder—it will come out in fragments, and we will keep this tube in for a longer period of time.

The reason he is having necrosis of his gall-bladder is because it was under tension in the first infection. I have seen the entire mucosa of the gall-bladder come away following infection. You could pick it right out after opening the gall-bladder. I recall my first case of this type. The patient had three attacks. He had gangrene of all coats of the gall-bladder, but the peritoneum was restored and the mucosa sloughed out and came away.

We have colon bacilli present here, and, therefore, we are in danger of having a colon bacillus infection of the cellular tissue. We mop the areolar tissue with a 1 per cent. solution of lysol, as it seems to have a good effect on colon bacillus contaminations.

In connection with gall-stones: 98 per cent. of the weight of all gall-stones is made up of cholesterin; over 99 per cent. of all gall-stones originate in the gall-bladder; a fraction of 1 per cent. of gall-stones originate in the intrahepatic biliary channels. Many of the gall-stones found in the liver are transmitted to the liver from the ducts by having obstruction below caused by a large stone. Small stones following it into the common duct and not able to pass it are forced up into the hepatic duct and back into the liver. Ninety-nine per cent. of gall-stones originate in the gall-bladder, and always as the result of infection. The most common place, therefore, where we have disturbance from stones is in the gall-bladder; the next is in the cystic duct; the next, in the common duct.

It is estimated that about 10 per cent. of all adults have gall-stones. It is further estimated that only about one in ten of these has manifestations of the presence of stones. These symptoms consist in manifestations of the presence of a foreign body in one of the positions I have mentioned. When these remain in the gall-bladder undisturbed, the rule is by secondary infection that they give little or no trouble. As they develop they give trouble in many ways. First and foremost is the disturbance of their position, so that they are dislodged or displaced as they attempt to pass into the neck of the gall-bladder and out of the gall-bladder into the ducts. Second, disturbances are produced by the secondary deposit of infective material around the stones or on the surface of the gall-bladder, which is irritated by the stones. Third, disturbances are produced by the increase of secretion from this infection, the secretion forcing the stones out or carrying them out with it through the neck of the gall-bladder into the cystic duct. They are carried on from the cystic to the common duct and from the common duct into the intestine.

When they are not infected and impacted in the gall-bladder, the way in which they produce trouble is by their mechanical presence only. When they become impacted in the gall-bladder they produce manifestations by the irritation that they cause and the evidence of absorption from the infection that occurs in the gall-bladder. That is great or small, depending on whether or not the contents of the gall-bladder are held under high tension by the impaction of the stone in the cystic duct, as was the case here, or whether it is merely an infection of the gall-bladder with virulent microörganisms, which is associated with chills and fever, edema, and swelling of the mucosa of the cystic duct, and makes the patient very ill, with the manifestations of infection, or whether it is an infection and ulceration of the neck of the gall-bladder from the impaction of a large stone, or retention in the neck of the gall-bladder without infection, gradually increasing the quantity of seromucous material in the gall-bladder under pressure, and producing continued distention of the gall-bladder. Whenever there is present only mild infection, and a stone of this magnitude is impacted in the neck of the gall-bladder, there is always a tendency to enlargement of the gall-bladder. The symptoms of cholelithiasis then are in this order: First, pain; second, nausea and vomiting; third, local sensitiveness in the region of the gall-bladder; fourth, elevation of temperature if infection is present; fifth, the presence of icterus when the stone has passed into the common duct—never before. You can see, therefore, that the element of icterus comes into play only when the stone is in the common duct. The order of symptoms is practically the same as in appendicitis, except that the sensitiveness is in an entirely different position. In making the test for sensitiveness perpendicular percussion takes precedence over others. Place the middle finger at right angles over the gall-bladder, and strike it with some force. Why is it that when we have so much pus in the gall-bladder, as we do from excessive infection, there is not a higher temperature? Because the fundus mucosa of all viscera has a meager supply of lymphatics. One rarely finds a temperature of 103° F. with septic cholecystitis, because there is so feeble a supply of lymphatics in the mucosa of the fundus of the bladder. That is an entirely different state of affairs from what exists when a stone passes into the cystic duct. There may be a small stone in the cystic duct, with retention and ulcer and pus present, with the temperature running up to 103° to 105° F. once or twice in the twenty-four hours, and continued for months, associated with chills, and resembling many of the types of ague, and still the patient may never have jaundice or a colic. The stone may pass on into the common duct, and because of the presence of the stone in the common duct and the existence of infection there occur elevation of temperature, chills and fever, and icterus.

Why is it that there occur such pronounced elevation of temperature, chills, and fever with stone in the cystic duct and in the common duct, and so rarely high fever or chills when the infection is in the gall-bladder? In the latter case there is an enormous quantity of pus—even to half or three-quarters of a pint, while in the former, two-duct types one may find only three, four, or five drops of pus around the stone. The reason for that is that the stone has abraded a narrow channel rich with lymphatics. You have just the same conditions with the presence of chills and fever and infection with a stone there that you have when the stone is in the ureter, in the presence of infection, and that is why chills and fever are so common in these two particular positions, and that is why infection in this particular position is so dangerous.

Kehr, in the early history of gall-bladder work, did a large number of choledochotomies in the presence of icterus and infection. Kehr found that when he operated in the presence of chills and fever and jaundice practically all his patients died from the infection and the icterus associated with it. When he operated on patients who had chills and fever without icterus, and with the stone in the cystic duct, only a small percentage of the patients died; but when he operated on patients with a temperature of 102° to 103° F. with infection of the gall-bladder, he practically lost none of them. The three different positions in which gall-stones lodge and the nature of the attack led Kehr and others to avoid operating on patients in the acute infective stage in the presence of jaundice.

I was led to operate on this man today because he had no jaundice. He had a stone impacted in the gall-bladder cervix for eight or ten days, and it would be only a short time, if the stone is of moderate size, before it would reach the common duct, infection would take place, and the patient would be exposed to all the dangers that go with such a condition. I operated in the acute stage because I was able to differentiate that his infection occurred in the gall-bladder, with a low temperature and a high leukocyte count, in contrast to what we would have had if the stone were down in the cystic duct or in the common duct, and in the absence of jaundice. We avoided the danger of perforation of the gall-bladder from that stone, which was impacted in the neck and might finally have ulcerated through, and the patient would have had a pericholecystic abscess which would hazard his life. Now, we have avoided the superlative degrees of pathology in this man's case and all the superlative dangers by a timely operation with a differential diagnosis as to where the lesion was.

The type of chills and fever occurring with small stones impacted in the cystic duct makes it difficult to diagnose in a differential way. Perhaps I can do no better here than to cite the history of one particular case which I saw in consultation. The man gave this history: He had had ague—chills and fever—for eleven years. He had been to all climates of the world to get rid of the ague. He was a wealthy man, and was able to go wherever he desired. He went to this place and to that place and finally landed in Pasadena, California. There he began having chills and fever of an irregular type. He had them at first once a day, then twice a day; then every second or third day. He went on in this way for some eight or ten weeks. He was losing ground all the time. He was seventy-eight or seventy-nine years of age. The chills were followed by a very high temperature—as high as 105.5° F. He never had a history of colic; he never had a history of jaundice; he had no history of trouble in the region of the gall-bladder. He had had innumerable examinations made for the plasmodium of malaria, and they were all negative. The only symptom he had was sensitiveness on deep palpation. You could hook the finger under his costal arch. He was a long, lank, lean Yankee, had no fat on him, and when he inspired with the finger hooked beneath the arch, it hurt him severely. The diagnosis was made of infection of the gall-bladder, and he was told that he should submit to an operation. He went to the hospital and I operated on him. He had a very small gall-bladder—not larger than the two distal segments of my finger. It was embedded in a large mass of adhesions. There was no stone in it.

It was a little pocket of pus surrounded by an enormous mass of cicatricial tissue. We split it open down into the cystic duct, but we could not put in a drain because the lumen was not big enough. We sewed a rubber tube with silk over the cut edge so as to prevent a closure of the opening and fixed it there. The next day he had a terrific chill, with a temperature of 106° F. He recovered and is alive today. The operation was done many years ago, and he has never had another chill. This was the last of this ague. Drainage was continued for several months.

I have cited that case to show you how high the temperature can be and how long the trouble can continue without the patient ever having a colic, without ever having a pain in the neighborhood of his gall-bladder. The only symptom in this case was sensitiveness. The temperature course was classic of cholecystic or cholangitic infection; that is, the temperature would go up to 104° to 106° F., remain at that point for a few hours, drop suddenly to normal, stay normal indefinitely, and do the same thing, up and down again, so that it was not the temperature-curve of sepsis, but the temperature "angle," which I described in Sajous' work when I wrote my article on gall-bladder infection. I described it as the temperature "angle" of cholangitic infection. It is classic in its appearance. The only instance in which you find it otherwise is in the infections which occur in infected erectile tissue or angiomata, as in the corpus spongiosum and corpus cavernosum, in the venous angiomata of children, and infections in the portal circulation.

We have come to recognize the fact in our modern work that where cholecystitis is caused by a cholelith, the disturbance is primarily in the gall-bladder. We are getting a smaller and smaller number of superlative degrees of pathology in cholelithiasis, and we are able to make a diagnosis, in a great percentage of cases, without the presence of jaundice at any time in the course of the disease. Years ago, in checking up 100 of our cases, we found that in only 14 of them did jaundice occur at any time in the history of the disease, so that you must make your diagnosis in 86 per cent. of the cases without the aid of jaundice.

When the diagnosis of cholelithiasis is made, then is the time to operate. Do not postpone operation and hope that the case will go on without producing further trouble, because these cases go on until they produce a lesion that greatly or materially hazards the life of the patient. Therefore, immediate operation after the diagnosis is made, or immediately after the acute attack passes over, is the time of election for the operation, because it is a procedure of little danger to the patient.

An examination was made after two weeks with an electric cystoscope. It showed that a small stone was present in the hour-glass contraction zone, where the large stone had formerly been lodged. The cystoscope was pressed on to this to the round ligament; a hook passed through the cystoscope to the stone. The stone was rotated and jammed against the edge of the cystoscope, and by this means it was extracted from its position.

The drainage was removed at the end of five weeks. The gall-bladder mucosa had resumed a smooth, glistening appearance, in contrast to the trachomatous appearance which had been present at the time of operation. The smooth condition of the gall-bladder is our guide in removing the drainage-tube.

TYPHOID SPINE

HISTORY

Physician, male, aged twenty-seven, single; has lived in the East all his life. Grandfather died of tuberculosis of lungs fifty years ago. One cousin has tuberculosis, but patient has never lived with him. No malignancy in the family. Two brothers and one sister living and well. Never had any venereal infection. Had measles at three years, following which had some trouble with both ears—a double otitis media. The left healed completely, but the right showed discharge intermittently until after the recovery from typhoid fever in December, 1910. Thus the chronic otitis media continued over a period of twenty-three years. An acute attack of right mastoid involvement at the age of twelve, and again after patient had been swimming during the summer of 1909 or 1910. Nasal diphtheria at age of eight years, which patient thinks aggravated the ear trouble. At age of fourteen had a fall, at which time it was thought skull was fractured at the base; there was a marked motor aphasia, which continued for three weeks, and some impediment in speech for two or three months; at the time of fall there was a copious bloody discharge from right ear.

Present Trouble.—Dates back to time when patient was seventeen years of age, during the time that he was still having the intermittent discharge from the right ear Patient noticed one day, while playing the piano, that he had pain in the metacarpophalangeal joint of forefinger of right hand. This was December 15th, and pain, "*redness*," and swelling did not disappear for about nine months, when the doctor immobilized the finger. *x*-ray showed thickening of the metacarpal bone under the periosteum. About five weeks after this finger trouble patient noticed, while skating, that "one" hip was sore on motion and caused a limp when walking. No swelling, no chills, no fever. Patient was in bed about one week, and upon getting up pain in both heels appeared. A trip abroad and three weeks' treatment at the baths in Italy gave no relief from the "rheumatism."

Went to New York and was treated with orthopedic shoes by a surgeon; some improvement in heels. The doctor said it was "flat-foot." Treatment for a long time.

Four and a half years after onset pain appeared in both hips. Pain was severe when first beginning to walk or run, but "wore off" after exercise. For three years following the pain in heels and hips was marked only upon severe exercise. While serving in hospital, doing heavy lifting, etc., patient noticed a dull, aching pain in his back, most marked in the morning, and disappearing during the day. Leather jackets, braces, hip supports, etc., were advised by several doctors.

Typhoid fever December, 1910; sick four to six weeks. Positive Widal and positive blood culture. Spinal trouble was greatly aggravated by the fever, and patient was subsequently treated for typhoid spine. At present had no pain in heels nor fingers or any other joints. Spine is painful upon exertion, as are the sacroiliac joints. Has sensation of loose joints in hips, which is overcome by pressure on hips.

Examination February 27, 1912: Has pubic and suprapubic varicosities mostly on left, showing that the deep iliac veins are closed. This condition has existed since his typhoid fever. Thigh (left) larger and skin soft. Lumbar spine and sacrolumbar articular and cervical vertebræ are fixed. Edema in left leg. About 80 per cent. of spread of limbs when flexed at right angles.

Wassermann test: Wassermann serum reaction for syphilis negative, but there is enough inhibition to hemolysis to make the presence of some abnormal condition probable.

Dr. Charles L. Mix: We have here a case of typhoid spine which Dr. Murphy has asked me to show you this morning. This young man had typhoid fever some time ago. He is a physician here in attendance at Dr. Murphy's clinic. The patient's attack of typhoid fever was subsequently followed by a typhoid spine, which is probably due, as these cases usually are, to the residual effects of the original infection. It is just as rational to suppose that the residual effects of the original infection can be aided by vaccine in a case of typhoid fever as it is to suppose that they can be aided in a case of streptococcic or staphylococcic infection. Consequently it has been thought wise to give him the benefit of vaccine treatment, and we are going, therefore, to give him this morning, subcutaneously in the arm, 500,000,000 dead typhoid bacilli. In ten days we will give him 1,000,000,000, and ten days after that another billion. If, at the end of three weeks' time, he still has further trouble, we will perhaps be justified in giving him a fourth dose of 2,000,000,000 bacteria.

DR. MURPHY: Will you give us the present status of typhoid vaccine as a prophylactic at this time?

DR. MIX: The results in the United States army have been exceedingly good in the prevention of typhoid fever by vaccination. I do not know whether you are familiar with the general subject or not. Vaccine prophylaxis for typhoid was first introduced extensively at the time of the Boer War. It met with a more or less indifferent reception, by reason of the fact that it was impossible for Sir A. E. Wright to follow up the end-results, so that at the end of the Boer War it was an open question as to whether there was real good done by it. It was abandoned temporarily. Subsequently, however, in England, about seven years ago, it was reintroduced by Sir W. B Leishman, and at that time it was used a good deal more successfully, especially in the English army in India, where the results obtained were very excellent. It then began to be experimented with in our own country. The first experiments of any note were made since 1908. In June, 1910, a small battalion of engineers stationed at Washington were ordered into the field. This group numbered about 120 men. Of them, over 90 had been vaccinated, and none of these acquired the disease, whereas of the 25 unvaccinated men, 6 subsequently developed typhoid fever. The last great experiment was made in the recent assembly of the army at San Antonio at the time of the Mexican rebellion, and on that occasion it was deemed advisable to make the matter a compulsory one, and each soldier who went to Texas was given the vaccine. The result was that only two or three cases developed in several thousands of the troops, showing conclusively that the prophylactic value of the vaccine is very great indeed.

I am very much interested in the general subject, as all internists are. Last September I developed typhoid fever myself, and thought I would try a little of the prophylaxis upon my own body. I went upon the theory that if the typhoid germs cannot be obtained from the blood until about five to six days after the onset of the fever—in other words, if it takes five or six days to bring about typhoid bacteremia, and if it takes only twenty-four hours or so to get the effect from the autogenous vaccine, it ought to be possible, in the first two or three days of the fever, to get ahead of the infection, the same as is done in the case of hydrophobia. The stage of incubation is sufficiently long in that disease to enable the physician to get ahead of the infection in the treatment of the patient. Why might it not also, in the very early cases of typhoid fever, be possible to get ahead of the infection? I, therefore, took a good-sized dose, 250,000,000, on the third day of the fever. The result was very interesting. I ran through a total number of twenty-one days of typhoid; I had a normal temperature several times during the course of these twenty-one days, although I gave a prompt leukopenic and a prompt Widal reaction, and I had the complications which are typical of typhoid fever, such as a phlebitis of the left external saphenous vein, which almost of itself is enough to establish a diagnosis of typhoid fever in a fever of that type running twenty-one days. Of course, I attributed the partially afebrile course to this vaccination.

I think that the typhoid vaccines can be used with advantage, provided they are used *before the stage of invasion of the blood* by the germs in any amount. I think, however, that it is perfectly useless to attempt to introduce the vaccine after that stage, because by that time blood cultures are positive. If results are to be obtained, they must be obtained before a generalized infection of the vascular apparatus has taken place.

DR. MURPHY: You are giving this man an injection, today, in what part of the body?

Dr. Mix: I shall give him 500,000,000 in the left arm. It does not matter, of course, where the injection is made—whether in the abdomen, the arm, or in the thigh. The arm is a very convenient place, and it will be given there in exactly the same way as one would give an ordinary hypodermic injection of morphin.

Dr. Murphy: Let the records show that Dr. Mix injected 500,000,000 dead typhoid bacilli subcutaneously into the arm.

I want to show you this young man with typhoid spine, and I want to call your attention to some of the things in connection with typhoid spine. This is not purely a case of typhoid spine, and that is a point I wish to accentuate. This boy has had repeated attacks of the "rheumatoid" type of spondylitis preceding this trouble. I say "rheumatoid," because it is supposed to mean more than rheumatism. It is only a nicer way of saying it; it does not mean any more. It means nothing.

The only place in which we could locate his primary infection was in his nose. He has had a continued catarrh. In one of our cases here the antrum was infected, then the frontal sinus; then the other antrum was infected, and then there were "rheumatoid" metastatic infections in many of the large joints. This boy did not have the sinus infections. An acute infection of his nasal mucosa was followed years ago by an infection in his spine. Now he comes here. He has no frontal or antrum infection now.

It is peculiar that from these mucous membrane infections we get these constitutional manifestations in the joints. We occasionally have them follow cutaneous infections, as we noted in the case of the man who had a furunculosis, and in whom a typical furunculous metastatic perinephritic abscess appeared. The infecting microörganism in both cases was the same type of staphylococcus, showing how easily these metastatic infections can be traced to their primary source.

In the course of typhoid, inflammation in the vein does not appear until eight or ten days, as a rule, and often not until twenty-five days, after the onset of the disease. You do not get a typhoid deposit in the bone as a typhoid osteal abscess, nor do you get typhoid arthritis until in the later stages of the disease, usually after twenty-one, twenty-two, twenty-three, or twenty-five days. Dr. Mix had phlebitis much earlier than usual when he had his attack of typhoid. There was a definite reason for it. Dr. Mix was compelled to remain out of bed on account of the intense pain in his back. He sat up in an easy chair much earlier than he should have done, and consequently he had to pay the price. That was the element in producing phlebitis so early in Dr. Mix's case, because we know that phlebitis occurs late in the other cases, and the other patients are practically always kept in bed, and are not permitted to get out of bed until after a certain length of time has elapsed.

Question (To Patient): How much trouble did you have with the spine or with the joints before you had typhoid?

Patient: I had considerable trouble two years ago. I was working pretty hard, and lifting was a good part of my work. I noticed I was stiff. It was hard for me to turn over in bed. I was stiff in the morning, and it was hard to straighten out. After I once straightened out and started on my work I limbered up, and I did not notice it again until I was in bed at night.

Dr. Murphy: When you began to work, how long was it before you noticed the first trouble with the back?

Patient: It preceded my work.

Dr. Murphy: The work accentuated the trouble in the back?

Patient: I think so.

Dr. Murphy: You do not know whether your spine was fixed at that time or not?

Patient: No.

Dr. Murphy: His spine is badly fixed, yet it is questionable whether it is ankylosed or not. The spine may become just as fixed as it is in this patient in the acute process without ossification of the intervertebral cartilages and without ossification of the perivertebral connective tissue. When the disease has advanced, as was shown beautifully in a specimen which I have in my office that came from Vienna, you not only have complete ossification of the intervertebral cartilages, but periosteal ossification, or ossification of the deep fibrous ligaments which connect one vertebra with another. It is typical of spirochetic disease, and is known as a Charcot joint, with the production, as well as destruction, of bone.

This patient has a negative Wassermann, so that we have here two conditions: (1) Involvement of his spine preceding his typhoid; (2) exaggeration or an increase in the spondylitis by an infection from his typhoid, and probably a second deposit of the typhoid bacilli in this entire zone.

In the management of this case we have outlined a definite plan: First, to neutralize the typhoid element by the injection of a large quantity of dead typhoid bacilli; second, after that is stopped, if we have no place where we can get an autogenous vaccine, we will use pyocyaneus vaccine. The pyocyaneus vaccine has the effect of increasing the number of lymphocytes and increasing the number of leukocytes generally. It also has a decided beneficial influence on this class of cases.

The best influence we get, however, is from the injection of autogenous vaccines.

We had a man under our charge who had his primary infection in his bladder. He came in with a typical "rheumatoid" arthritis. He had colon bacilli and Staphylococcus albus in his bladder, associated with a severe cystitis and a continuous discharge of pus, following the removal of the prostate.

At the end of some weeks one joint after another became involved, until practically every joint in his body was invaded by the infection. He was put on autogenous vaccine, first 50,000,000, but that had little effect. We increased the dose to 100,000,000, to 150,000,000, to 200,000,000, and then he commenced to get a reaction and improved.

The patient was sent to us with the idea that there was no hope for him. His case was one of typical "rheumatoid" arthritis, and it was thought that the process would go on until finally all his joints would become involved and ankylosed, and he would be completely incapacitated. We believe that, with the treatment he has received, he is going to make a full and complete recovery—that is, the autogenous vaccines are doing the work. He is taking, in addition to the autogenous vaccines, treatment which aids his elimination; he takes hot baths and electric baths. He is in Hot Springs now. We believe that the greatest benefit is to come from the use of the autogenous vaccines. He has also had from three to five injections of 2 per cent. formalin in glycerin in all his joints.

One cannot escape from the fact that the typhoid vaccine has had a great influence in a prophylactic way. Its specific influence is as great as that of antitoxin in diphtheria. Again, one cannot escape from the fact of the positive prohibitive effect of injections of the antitetanic serum. We have not had a single case of tetanus occur in this hospital since we began using immediate injections of antitetanic serum, running over a very large number of cases of different types of crushing injuries, and the same class of injuries from which we formerly had one or two cases of tetanus every month. The reason internes in this hospital have no experience with tetanus is that they do not know what tetanus means from observing its evolution in cases treated, except when a patient is brought in who had tetanus before the antitetanic serum had been used.

We give the antitetanic serum at the time of the injury. It has little, if any, effect once the disease has developed, and it is not intended to cure. It is intended as a prophylactic, and not as a curative method of treatment.

A few fatal cases of anaphylaxis are recorded in the history of antitetanic serum injections. Asthmatic patients should be questioned closely concerning the etiology of their "asthmatic" spells.

EXTRADURAL HEMORRHAGE FROM TRAUMA: EXCISION OF THREE AND ONE-HALF INCHES OF DURA

HISTORY

Male, twenty-five years of age, entered hospital March 11, 1912, because of symptoms resulting from a blow on head thirteen years ago.

Family history negative. Was always very healthy until the age of puberty, when injury occurred.

Present Trouble.—In February, 1900, when thirteen years of age, he was struck on the left side of the head, 4 inches above the ear, by a strong, fifteen-year-old boy, using a piece of board 4 feet long by 6 inches wide. His father thinks that he was rendered unconscious for five or ten minutes. He was carried to his home. He had no headache. A surgeon who saw him sewed up a small scalp wound and pronounced the injury not serious. Next day the child was apparently as well as usual.

He was always a bright, active, talkative boy. He never stammered, nor was he unusually quiet or backward in any way. Following the injury he attended school and carried his work with credit. Seven months after the injury his mother noticed that he was becoming more nervous, more easily excited, and more irritable than usual.

During 1902, two years after the first injury, he fell backward from a cart on which he was riding, falling a distance of 4 feet, and striking on the back of his head on the unpaved roadway. He was rendered unconscious for about two minutes, but was able to climb back into the cart and ride home. The scalp was not wounded, and no ill results were apparent. This second fall seemed to affect his nervous condition and change his disposition.

He became irritable—very much so at times. He was easily excited and talkative. His mother noticed that while talking rapidly and easily, as he always did, he would occasionally halt in the middle of a sentence as if for a lost word. This continued and very gradually became worse, and, unlike his natural self, he became embarrassed easily. His brother discovered, while at school, that he became slower and more labored in his answers. Finally he could not keep up in his work and quit school altogether.

During 1904 his right hand and wrist became weakened, and he could not use them as usual, though at no time did the hand become completely paralyzed, nor were there any movements which he could not make if he continued the effort long enough. No atrophy has ever been apparent.

Four years ago he began to have trouble in swallowing, and while eating would choke frequently. The face seemed more flaccid on the left side, and when he smiled the angle of the mouth would pull to the right somewhat. Saliva often would drool from the angles of the mouth.

At about the same time that difficulty in the face began his father thinks his right toe and leg dragged slightly as he walked. At no time did this become pronounced.

He has complained of a giddy, dizzy sensation in his head often, but he never has had a headache, vomited, or become unconscious, nor has he ever had convulsions or muscular contractions of any type. During the past two years he seems more irritable, and at times is unmanageable. At these times he becomes angry, and then he moves and talks better than at any other time.

His doctor states that eye-ground examination has repeatedly proved negative. Eye reflexes are normal. Sensation is perfect over the entire body.

Laboratory Findings.—Urinary examination proved negative.

COMMENTS AND OPERATION

Dr. Murphy: Dr. Mix will give you some points in connection with the history of the case of this boy.

Dr. Charles L. Mix: An examination of this patient gave approximately the following findings, from a neurologic point of view: As to the reflexes: His tendinous reflexes, his bicipital and tricipital, and those of the extensors of the forearm were all exaggerated; the patellar and the Achilles jerks were also exaggerated. The pupillary reflexes, both as to light and accommodation, were normal. The epigastric and abdominal cutaneous reflexes were absent; the plantar reflexes were present and normal; there was no abnormal reflex on the part of the upper motor neurons, that is to say, there was no Babinski, Gordon, Oppenheim, or Chadwick reflex to be obtained. As to sensory disturbances: I put a sharp-pointed pin through the skin of his back without much manifestation of pain on his part; but on account of a defective mentality, we are unable to say whether there is any real disturbance of sensation or whether there is rather a disturbance of apperception. He had, on the tip of the left forefinger and of the left third finger, burns from matches, which might possibly suggest syringomyelia, but when one examines him further he is certain that these burns were accidental by reason of the lack of appreciation rather than by reason of any interruption of any of the conductivity of nerve impulses. He also has a scar from a wound on his left forearm, this lesion having been sewed up by Dr. Brooks without much manifestation of pain by the patient. Owing to his mentality and to his inability to speak we could not, by the simplest language, induce him to indicate to us anything about his muscle and joint sense, his tactile, temperature, and pain senses, so that this question of sensory disturbances is left, so to speak, unsettled.

As to atrophies, a very important neurologic sign: In looking over his scapular muscles, the suprascapulars and infrascapulars and the rhomboidei, we found no atrophies whatever. Both the right and left trapezius muscles were well nourished. His humeral group, triceps and biceps on both sides, are of equal development. As to the forearm group, both the extensors and flexors are developed alike on two sides, and there is no atrophy of the muscles of the lower extremities or the trunk, the back muscles being fairly normal except for a mild degree of scoliosis.

As to motor disturbance: He has spasticity of the right arm and of the right forearm and right hand, and he also has spasticity of his lips and of his tongue. Besides these *spastic* disturbances, there is a *paretic* disturbance of some of the muscles. He has spastic paresis of his right arm, forearm, and shoulder-girdle group of muscles, and of the right side of the tongue and of the facial muscles about the mouth. He has, however, the power of grasping. His lower extremities show nothing; they are absolutely normal, without spasticity or paresis. He walks perfectly well, without spasticity, paresis, or ataxia. He has apparently no intention tremor in his hands. When you have him make the test-tube test, that is, pour water from one tube into the other, he does it without spilling a drop of water. It is not truly an intention tremor, but a spastic tremor, when carefully analyzed. It is on account of that spasticity that he cannot button his clothes and cannot dress himself.

He is depressed, he is emotional, and he cries easily. His finer sensibilities have gone by the board to a slight extent; he wets his trousers without being particularly disturbed about it.

There are some negative points. There is no scanning speech, no nystagmus, no Romberg sign present, no ataxia of the lower extremities, and no ataxia of the upper extremities.

As to the conclusion to be reached in this case, the first question is this: Is the spinal cord affected in any way? If it is, we have to entertain the possibility of multiple sclerosis. There is no sign of involvement of the spinal cord from top to bottom. There is no atrophy in any part of the spinal musculature, and no disturbance in the anterior horn cells. There is no abnormality in the reflexes of the cord to indicate that there is anything the matter with his spinal cord. There is no loss of the bladder control—in short, nothing to which one can attribute his malady so far as the spinal cord is concerned.

The next question is, Can you charge up any of these things to his medulla? Is the disturbance a medullary one, or is it one that is far forward? When one finds a medullary lesion affecting the lips and tongue, suggesting, as does this case, the diagnosis of bulbar palsy, one ought to find, associated with deviation of the tongue, atrophy of the same side. But he has no atrophy of that side of the tongue; therefore there is no real reason for believing the disturbance of paralysis with deviation of the tongue and of the paresis of the lips, without atrophy, to be due to bulbar disease. Moreover, if the disease were really bulbar paralysis, with the twelfth and seventh nerves involved, why should the intervening nuclei escape ? Moreover, there is no vertigo. Most of these medullary cases show vertigo on account of involvement of fibers running up into the cerebellum through the cerebellar peduncles. He has none of these. The conclusion is that there is no medullary trouble.

There remains for consideration the cortex. With the history of injury which we have in this case, the most sensible diagnosis is that of subcortical cyst, which would easily follow such a trauma, and which would be responsible for the set of findings we have, or it may be due to a thickening of the dura over the motor area supplying the right arm and the speech-center. Since many of these cases of early trauma are later followed by cysts, the finding of a cyst would not surprise us when the operation is done.

Now, of course, in arriving at a conclusion, I did not have the benefit of an *x*-ray plate. An *x*-ray has been made since I examined the patient. The differential diagnosis, when I saw the patient yesterday, was between multiple sclerosis, bulbar paralysis, and organic disease of the brain cortex, and we decided in favor of the latter. With the *x*-ray plate I think there is additional reason for the conclusion that the motor cortex is diseased or compressed by dura thickened from trauma.

The next thing to do is to resort to trephining and to the clearing up of the situation, so far as it can be done; but, of course, if there be disturbance of the underlying cortex of any grave amount which has lasted over a course of twelve years, we cannot expect to have restoration of that cortex to the normal.

Dr. Murphy: What Dr. Mix has said gives us a basis for action, and the line of investigation we have carried out in this case should be carried out in all cases. Dr. Mix has had an exceptional experience with lesions of the nervous system, consequently his opinion is worth more than the surgeon's opinion in the diagnosis of these lesions, whatever they may be, and he has outlined the various symptoms to you in a very lucid manner. On that basis, and with the *x*-ray findings, there is only one conclusion to draw in this connection, and that is there is a possibility, if not a probability, of our being able to do something for this patient in a surgical way. This boy is becoming more irritable, so that it is very trying to his parents to put up with him. We do not know whether he has a subdural cyst or not until we have operated. We know, in the epileptic cases, after removing a portion of the indurated and adherent dura, it lessens the irritability of these patients often for months and even years, and that has been our plan of management in this particular class of cases.

You remember the case we operated on a week ago today. That man is now able to move his arm better than at any time. He is able to throw the arm around. His speech has improved materially, and yet it is only a week since we took out a cicatricial mass. He is better now than at any time since he received his injury, which shows the significance of removing the constant source of irritation.

The other type of lesion which Dr. Mix alluded to, thickening of the dura due to a primary pachymeningitis or to a subcortical cyst, comes on after injuries, grows slowly, finally attains considerable size, and drives so many of these patients into an asylum. It is one of the things that has been overlooked by surgeons and physicians until very recent times. You doubtless remember the splendid paper written by Dr. Dudley P. Allen, of Cleveland, two or three years ago, in which he analyzed 12 or 15 of these cases, following comparatively mild traumatisms, most of them not sufficient to render the patient unconscious, this condition developing under the scar. This boy, I understand, has never had convulsions. Here we have a case for operation on the recommendation of the medical man as a probability—better, as a possibility—of doing something for relief. In this case there is absolutely nothing to lose. Death is preferable to his present condition. The boy is becoming very vicious in his thoughts and in his actions, and so pronounced are these things that he has to be separated from his family. At first there was

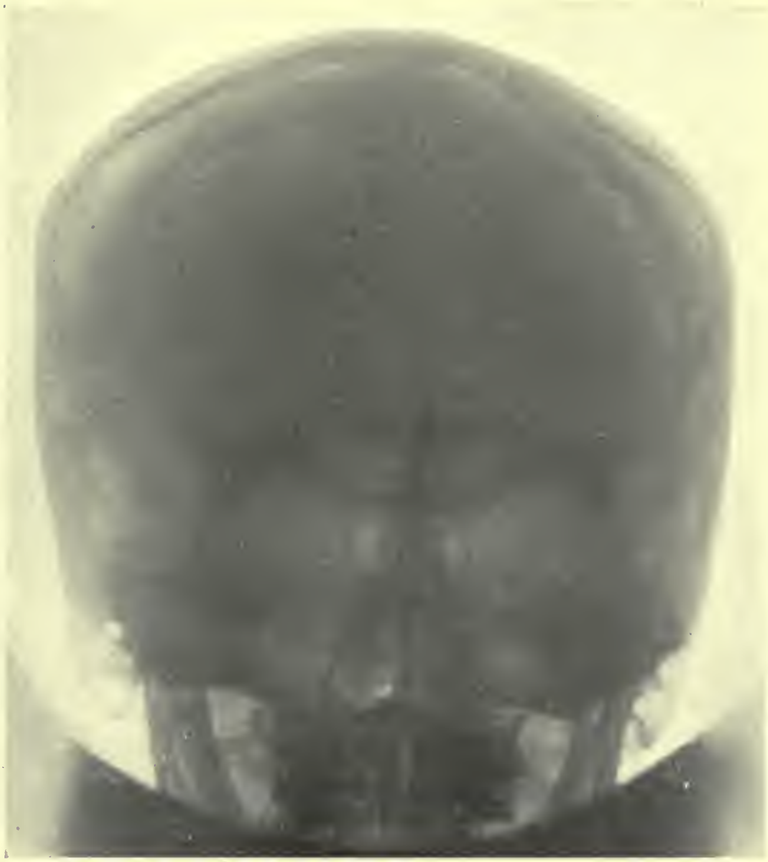


Fig. 82.—Skiagram showing apparent thickening of skull on the right side.

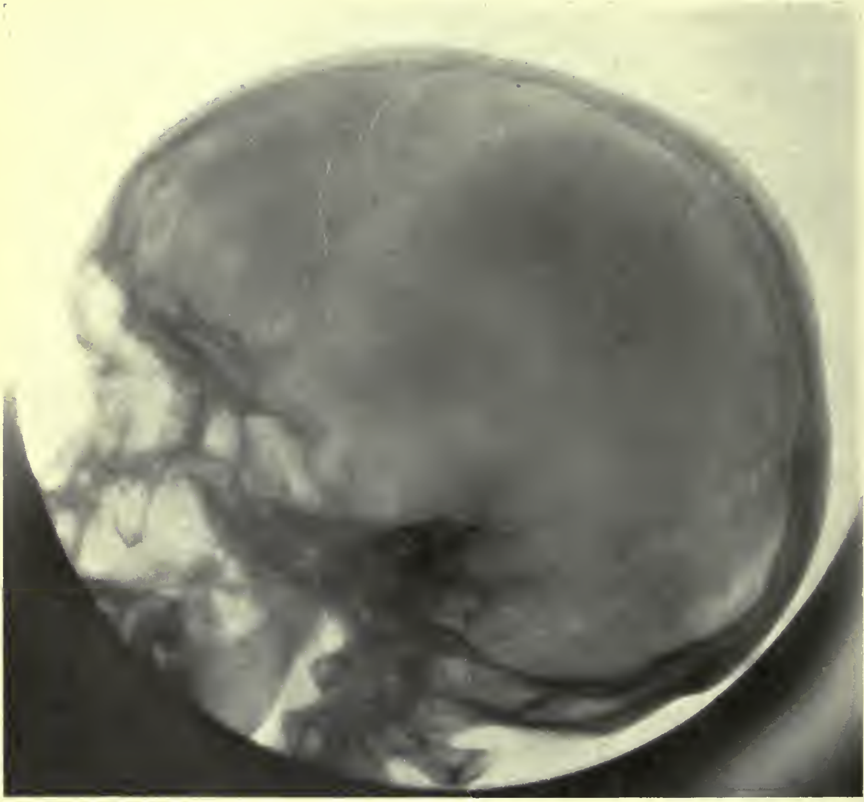


Fig. 83.—Skiagram showing shadow posterior to sagittal suture just below vertex.

none of that. Those things occasionally occur, but not often, in the epileptic, due to local irritations from a fracture.

We will make the usual exposure by making a U-shaped incision for the purpose of raising a skin, bone, and dural flap, 4½ inches broad and 3 inches long, from the occipitoparietal zone, so as to expose the Rolandic area. The hemorrhage is controlled by a rubber tube constrictor. The skull is opened with an electric trephine of Dr. J. M. Neff, and the electric craniotome, also devised by Dr. Neff. The skull is thick—very materially thicker on the anterior margin at the position in which he received the greatest force of the original injury. The bone does not fragment like the other bone, showing that its vascularity has changed. We are making this large horseshoe-shaped flap because any operation which will avail must avail by a large exposure. This deVilbiss instrument, I think, next to the Neff instrument, is the best instrument we have for this work. The advantage which the deVilbiss has over the Neff instrument is that it works more regularly. The instrument is of disadvantage because it requires two or three times the amount of work, and many more units of physical energy, on the part of the operator.

Now I have made this large horseshoe-shaped flap, and will narrow it near the lower end so as to control the line of fracture. We will not go down deep into the fossa, as we can separate the dura quite a distance below the line where we fracture the bone.

We detach the dura over the entire zone to which we wish to have this fracture expose the brain. Now we have a fairly good exposure.

There is no tumor mass to be felt. The brain is very soft right over his speech-center. See how that depression stays where I put in the handle of the scalpel! It is just where he was struck. We take out all that zone of thickened dura to stop that irritation. I cut that dura so that the margin of the dura comes inside the bony portion of the flap all the way around, so you can get so much decompression.

He probably had a subdural hemorrhage which gave him this softening that he has in this position, and it is a lesion of trauma, rather than a lesion from a constitutional condition, which was also our conclusion, and this accentuates the significance of the early management of brain traumas and what should be done with them.

For a great many years—not originally our own suggestion, but on the suggestion of that master mind, Dr. Fenger, who was so prominent in the last century—we have followed the plan of his original work. In every skull injury where the patient has been momentarily stunned to a degree to render his recollection indistinct, we have put the patient to bed, insisting on the maintenance of the recumbent position for a period of at least six weeks. By doing that we believe that we have secured better results than we did by the former plan of management, namely, allowing them to be around immediately after the effects of the injury had passed.

From our knowledge of the histology and pathology of the brain we find that in concussions and contusions it requires the brain a longer time to get its normal circulation restored than it does any other tissue, and for that reason, whenever the brain is concussed or contused, it demands a long period of rest to have repair take place with the formation of least connective or cicatricial tissue.

If you have a broken leg or a bone injury in any other position of the body where you can see the lesion, you endeavor to keep the patient quiet during the process of repair, so as to have the parts approximated as accurately as possible, and to get restoration of the tissue with the least intervention of newly formed cicatricial connective tissue. Wherever you have a large quantity of newly formed cicatricial tissue you have contraction; wherever you have contraction you have irritation, and this is particularly injurious in the brain. We believe in the plan of Fenger and have followed it for a long time. It requires a great deal of perseverance to control patients, and it requires a great deal of persuasion to show them they are in error if they endeavor to get about before the process of repair has made sufficient headway.

Every interposition of connective tissue is a detriment to the function of the organ or viscus after repair has taken place, so that it is an important factor in the early management of these brain cases.

What are the dangers that occur in connection with this type of injury, namely, the blow which this boy received on the side of his head, anterior to the middle meningeal artery, and high over the upper portion of the tip of the anterior branch of this artery?

The dangers are these: *First*, the effect of the concussion, an immediate communication of the disturbance to the cell circulation; *second*, a disturbance in the circulation of the brain or meninges, such as laceration of the middle cerebral or meningeal artery.

The average case of rupture of a middle meningeal artery has a typical cycle or course, and I cannot do better than mention here the clinical history in the case of a man who was playing polo. He was struck by the ball on the temporal region. It unhorsed him; he got up dazed, remounted his horse, rode about a bit, but did not enter the game. He went home; on his way he felt dizzy and had a headache. He was put to bed, and little attention was paid to him for an hour or two, when it was discovered that he was completely unconscious, and then, of course, the alarm was given, physicians were called, and a diagnosis made of rupture of the middle meningeal artery. The former plan of management for that was attempted—sending to the city for a doctor, as he was in a suburban town. The doctor came there with all haste, with all his instruments, and in the mean time the patient had gone into a profound stupor. Before this doctor saw him he was seen by a very distinguished doctor, who also believed the trouble would amount to nothing, because he was a little stupid in his responses, complained of his headache, and the doctor said, "I believe it is just a slight concussion and means nothing more." When the second doctor arrived with his instruments he did a trephining operation. By that time a large clot of blood had formed from the ruptured middle meningeal artery, and the patient never rallied from the operation.

I simply cite that to show you the cycle in the course of the symptoms. First, after the immediate shock, they get up and walk about, as a rule. They are fairly clear in their intellect, and get along nicely, and then gradually become unconscious. That is practically pathognomonic of rupture of the middle meningeal artery, either intradural or extradural.

All you need to do when you feel that the patient is going into a state of coma on account of rupture of the middle meningeal artery is to make a little incision under cocain anesthesia, salt solution, or sterile water analgesia, cut down on his external carotid, and throw a ligature around it. That stops the hemorrhage, and you are master of the situation immediately. The middle meningeal artery is a branch of the internal maxillary, which is a subdivision of the external carotid. It has a terminal circulation, so that one ligature stops the bleeding. You get him out of danger at once, and you have not hazarded the patient at all. It is a simple matter, and uniformly overlooked in the management of these cases. A diagnosis can practically always be made, as they all have some return of consciousness after the concussion. You have a contusion or depression at the site of injury, which tells you exactly what has happened. After ligating the carotid one can take his time to do the trephining.

Let the record show that there were no adhesions to the dura; that there was a large, softened area, as large as a bantam egg, just at the lower end of the fissure of Rolando; that a depression of the pia there left a concavity that it took several seconds to fill. There was no tension on this, and for that reason it was not thick. There was an increased vascularity in the veins over that zone and over all the zone from which the dura was removed. That a piece of dura, 3½ inches long by 2½ inches wide, was entirely excised; that this was not excised clear to the margin of the bony elevation—I left ½ inch of the dura on which the edge of the replaced bony flap could rest; throughout the entire circumference of the exposed zone there were no adhesions to the pia; that there was some inflammatory process produced within the brain substance itself around the speech-center which continued to a softening and degeneration of the brain following trauma; that there was an extradural hemorrhage from trauma which caused a cicatricial formation in the dura.

What we hope to secure here is not the restoration of function in this zone, but a suppression of the irritability, which is often obtained when we excise the dura; if we accomplish that, we will do well.

The patient convalesced without untoward symptoms. There was no material change in his general condition up to the time he left the hospital, five weeks later.

POTT'S FRACTURE

HISTORY

Patient, a girl aged fifteen, admitted to Mercy Hospital January 5, 1912. Family and personal history were negative.

Present Trouble.—On June 8, 1911, the patient stepped on a stone; her foot slipped and was forcibly everted. She fell to the ground and could not rise. The ankle and leg were much swollen, red, and intensely painful. The leg was placed in a fracture-box at once, and twenty-four hours after the accident a mold was applied and left on for six weeks. When it was removed the ankle was deformed and the foot was everted. The patient has been able to walk since, but limped. There was visible shortening of the leg. She could not hold the foot in a straight line with the leg.

COMMENTS AND OPERATION

Examination.—The proximal portion of the tibia was overriding internally the base of the internal malleolus, and the latter was directed downward and outward instead of backward and inward. The external malleolus was broken off and displaced outward and also upward on the outer surface of the fibula. A plumb-line drawn through the middle of the tibia passed just to the inner side of the articular surface of the astragalus, showing the outward luxation of this bone. There was a pronounced abduction of the foot. It was a classic superlative deformity from a Pott's fracture.

Operation.—The internal malleolus was exposed by an incision to the inner side of and parallel with the tendon of the tibialis anticus. The line of fracture at the base of the malleolus was clearly demonstrated. It was refractured a little higher up, and on a line directed upward and inward from the articular surface, as so to permit of sliding upward when the deformity was reduced. The external malleolus was then exposed and divided on a line more slanting upward and outward. The newly formed interosseous ligamentary attachments were freed, and the foot was displaced inward. The internal malleolus slid upward on the inner surface of the tibia, and the external malleolus was slipped downward on the outer surface of the fibula. The internal malleolus was then fixed with one nail driven upward and outward, and the external malleolus was fixed with two nails, so that the midline of the articular surface of the astragalus was held a little to the inner side of the midline of the articular surface of the tibia. The foot was dressed in an adducted position and flexed on the tibia. Primary union resulted. The mold was removed at the end of four weeks, when motion in the ankle was perfect and painless. Seven weeks after the operation the nails were removed. Complete result. Complete bony union with a little thickening of both the external and internal malleoli, due to the callus around the large, freshened bony surfaces made during the operation. The patient stands plumb on her foot, and has perfect motion on leaving the hospital, March 7, 1912.

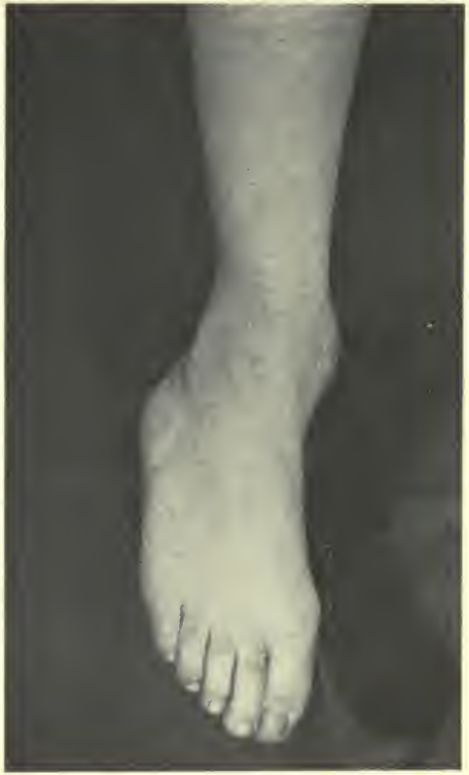


Fig. 84.—Pott's fracture. Photograph showing deformity before operation.

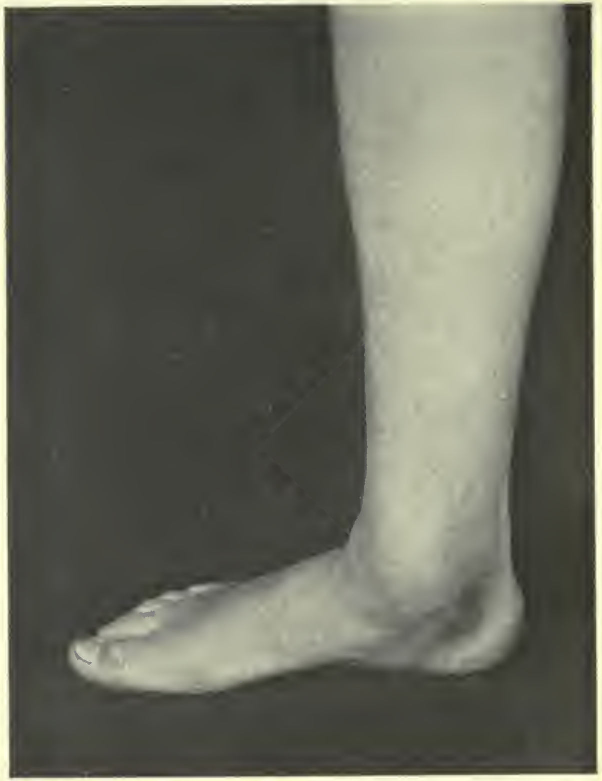


Fig. 85.—Pott's fracture. Photograph showing deformity before operation.

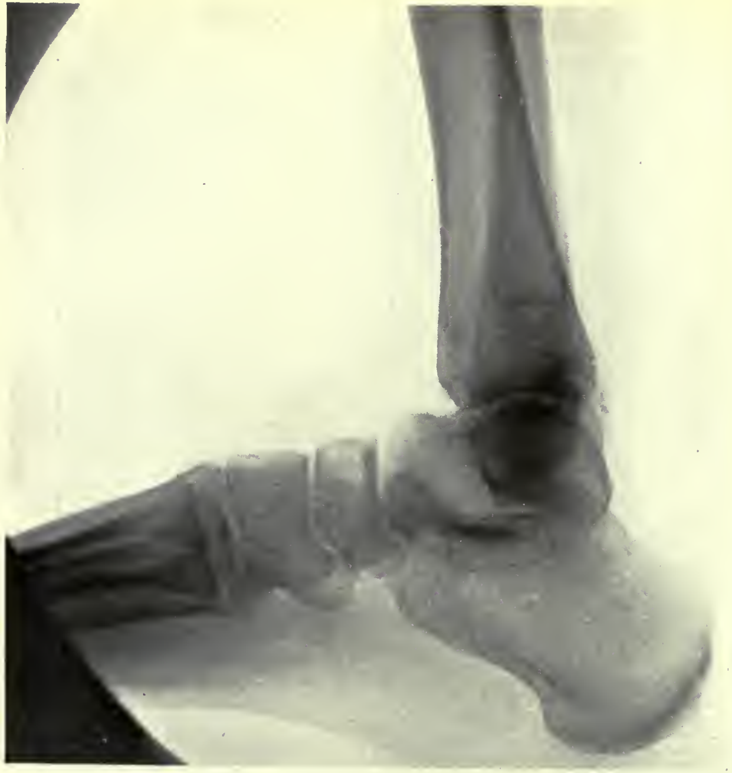


Fig. 86.—Pott's fracture. Skiagram showing Pott's fracture before operation.

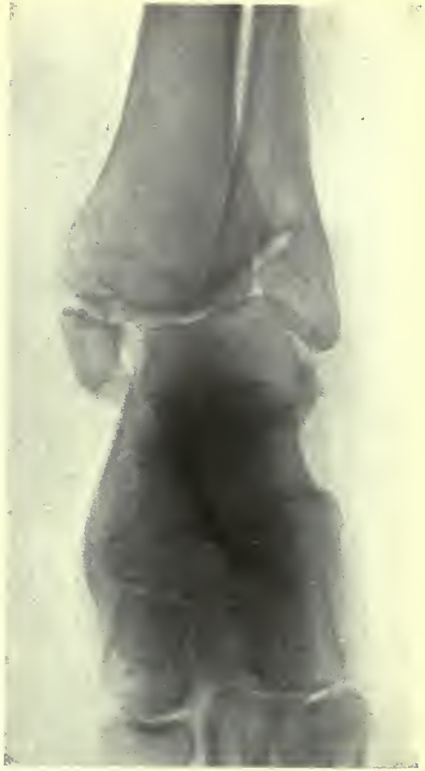


Fig. 87.—Pott's fracture. Skiagram showing Pott's fracture before operation.

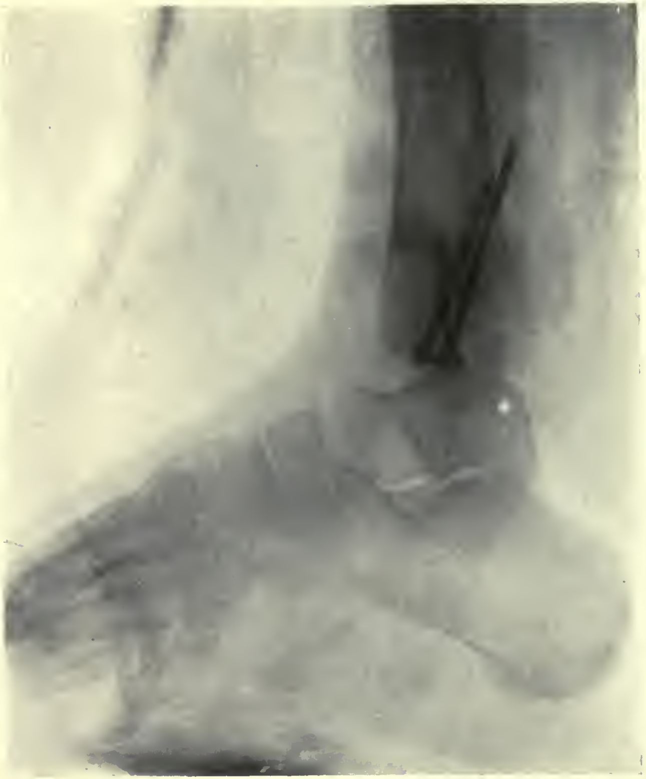


Fig. 88.—Pott's fracture. Skiagram taken through the cast two weeks after operation, showing fragments held in position by the nails.

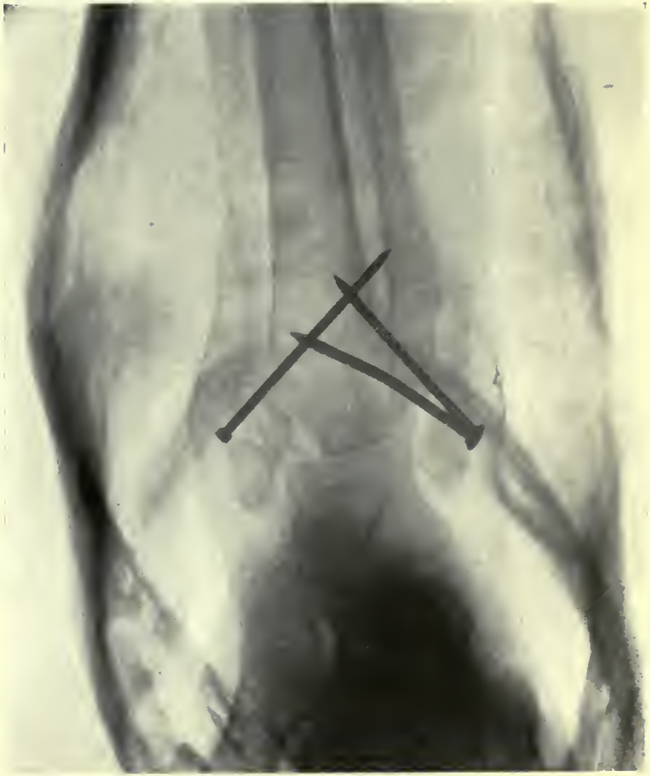


Fig. 89.—Pott's fracture. Skiagram taken through the cast two weeks after operation, showing fragments held in position by the nails.

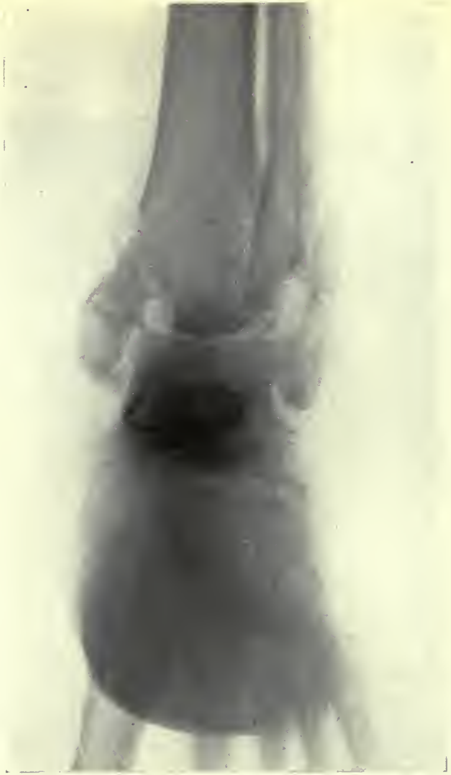


Fig. 90.—Pott's fracture. Skiagram taken after removal of nails two months after operation. Bony union between each malleolus and its respective shaft. Astragalus in plumb-line with articular surface of tibia.

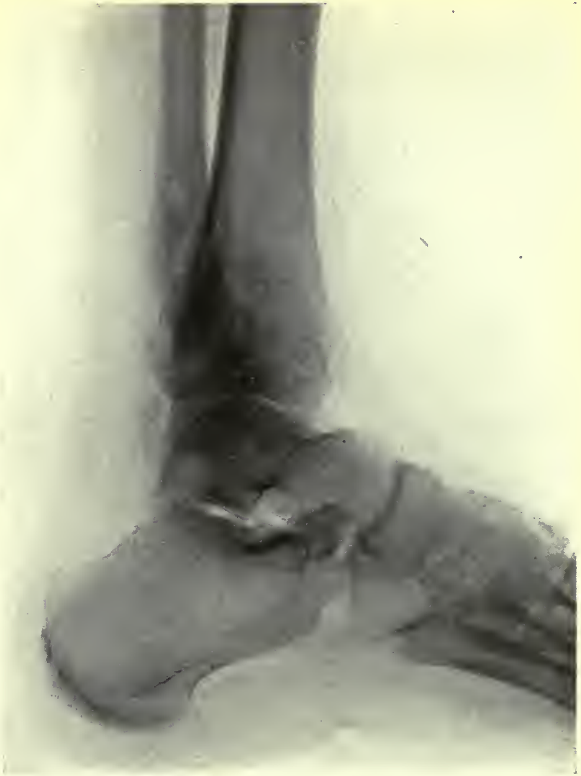


Fig. 91.—Pott's fracture. Skiagram taken after removal of nails, two months after operation.

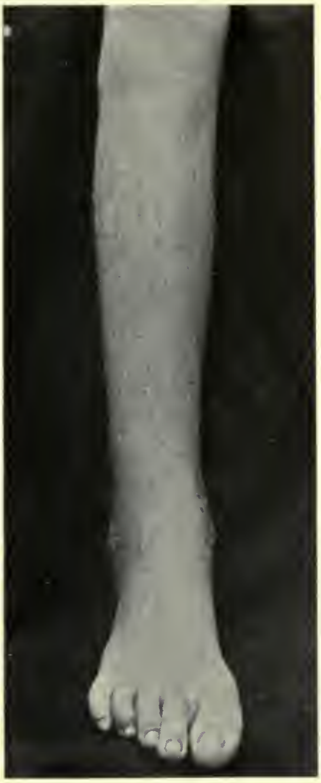


Fig. 92. — Pott's fracture. Photograph taken when patient left hospital, and showing results of operation. Symmetry restored.

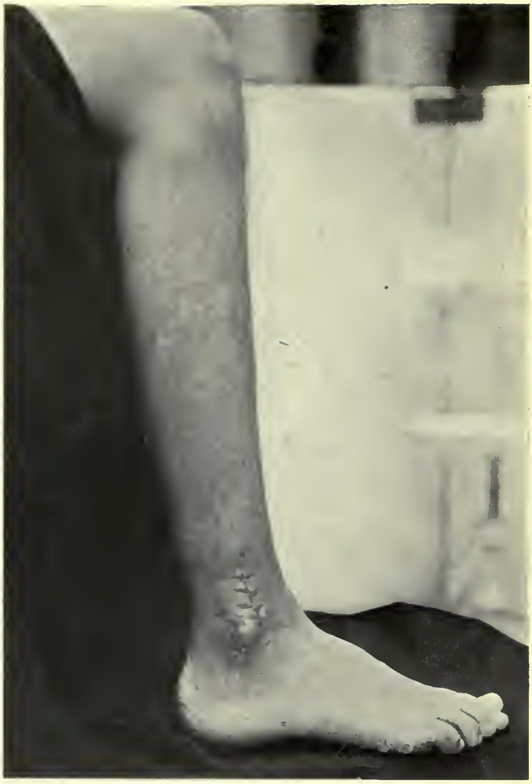


Fig. 93.—Pott's fracture. Photograph taken when patient left hospital ten weeks after operation. Symmetry restored.

FIVE DIAGNOSTIC METHODS OF JOHN B. MURPHY*

In the every-day work of a large surgical clinic, where there is maintained an aggressive spirit of progress, new ideas and new methods are constantly being tried, and deductions made as to their value. Those proving of no merit are soon discarded, but those of worth are adopted, and in time become a part of the regular routine procedure. The longer the accepted methods are used without showing signs of being fallacious, or without being superseded by better ones, the more weight is given them. This is true in the matter of operative technic, but it is still more true of diagnostic methods, and the latter, if possessing real merit, are of value not only to the surgeon, but also to the general practitioner.

In the service of Dr. J. B. Murphy there are in use five diagnostic methods which were original with him, and which have been used by him for a great many years. They have been thoroughly tried out, and have proved their unquestioned value as aids in the differential diagnosis of some of the pathologic conditions within the abdomen. Their reliability and simplicity of execution make them easy of application and increase their usefulness, especially to the physician in general practice.

The first is what has been called by Dr. Murphy the "fist percussion of the kidney," and is used to determine the presence or absence of an acute pathologic condition within that organ. As shown in Fig. 94, the patient, with clothing removed above the waist, is seated in an upright position on a stool, and is then instructed to bend forward as far as possible. With the patient in this position the examiner, from behind, places his left hand flat upon the back of the patient over the kidney of the one side or the other, care being taken to have the hand pressed firmly upon the back. The clenched right hand of the examiner is then brought down with considerable force upon the dorsum of the fixed hand (Fig. 95), and if an acute congestion, infarction, retention in the pelvis of the kidney, or ureteral obstruction exists in that kidney, the patient will cry out with the pain of the blow. It is analogous to striking a distended urinary bladder, and if the pathologic condition be present, the result will be decidedly apparent. A good sharp blow is used, but it is not necessary to use force enough to aggravate any diseased condition within the kidney. A control test is advised by first using the blow on the sound side and then on the suspected side.

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* [*Editorial Note.*—Dr. Murphy, in his clinical talks, constantly refers to the "five diagnostic methods" described in this article. In order that our readers may thoroughly comprehend these we print the article complete as it was published in the "Archives of Diagnosis" in January, 1910. The article was written by Dr. Guy G. Dowdall, at that time a member of Dr. Murphy's staff.]

A second method, and one depending upon the same principle as the preceding one, is used by Dr. Murphy in his examination for an acute infection of the gall-bladder, or acute obstruction of the cystic or common duct, with or without infection. The examiner, sitting at the right side of the recumbent patient, presses the tip of the second finger of the left hand, flexed at a right angle, firmly up under the costal arch at the tip of the ninth cartilage, as shown in Fig. 96. The patient is instructed to take a deep breath, and at the height of the inspiration, when the gall-bladder is forced below the costal guard, the flexed finger is struck forcibly with the ulnar side of the open right hand of the examiner, and if there be an inflammation or a retention in the biliary tract, the patient will announce that the blow caused him severe pain. One is again striking an overdistended or inflamed viscus that contains extremely sensitive nerve-filaments. Dr. Murphy calls this the "hammer-stroke percussion." The force used in striking the finger is well shown in the illustration (Fig. 97), for here one sees that sufficient force was used to sink the finger well up under the costal arch.

Another method, designated by him as the "deep-grip palpation," is somewhat similar to the above, and is also used in an examination of a suspected gall-bladder disease. It is based

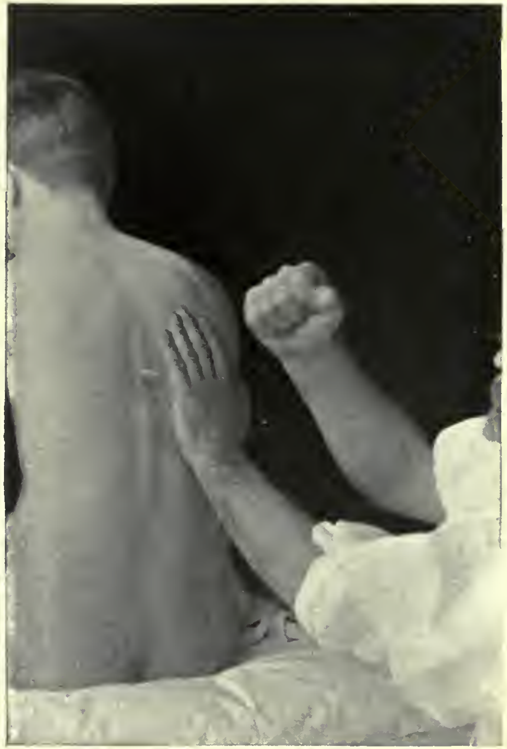

Fig. 94.—"Fist percussion of the kidney."

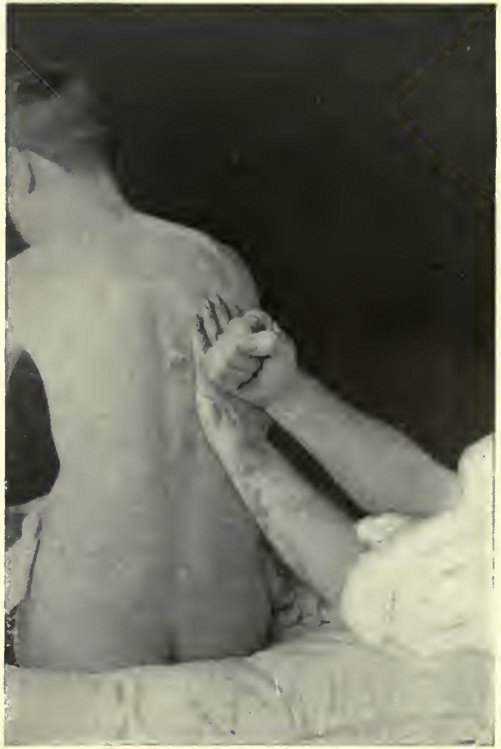

Fig. 95.—"Fist percussion of the kidney."

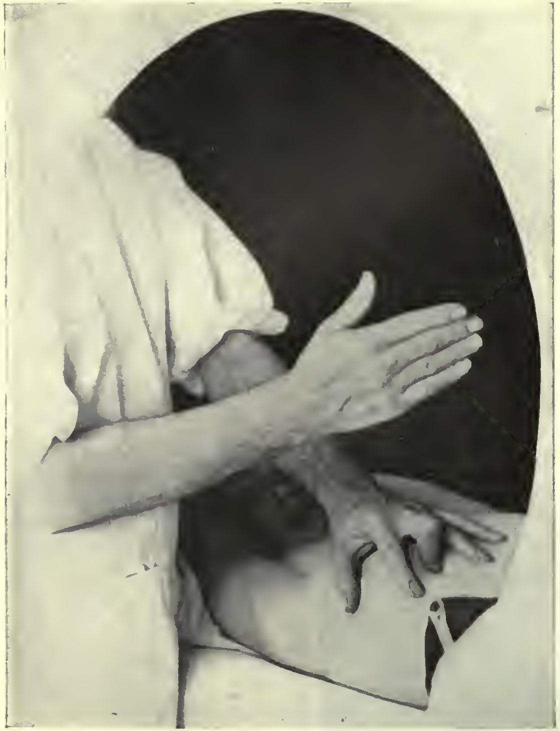

Fig. 96.—"Hammer-stroke percussion."

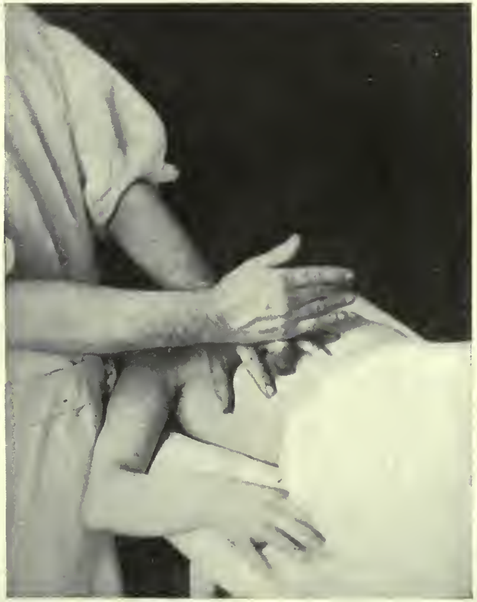

Fig. 97.—"Hammer-stroke percussion."

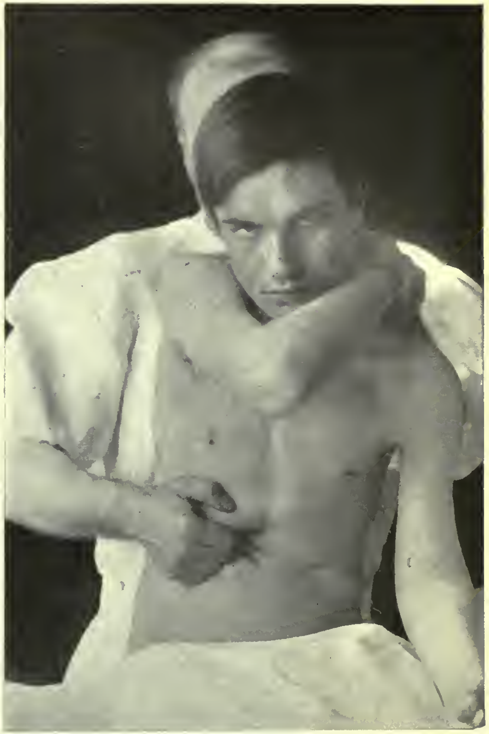

Fig. 98.—"Deep-grip palpation."

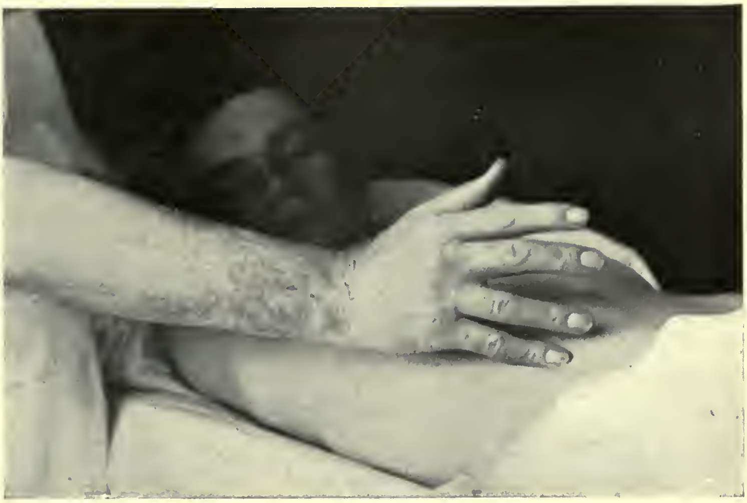

Fig. 99.—"Piano percussion."

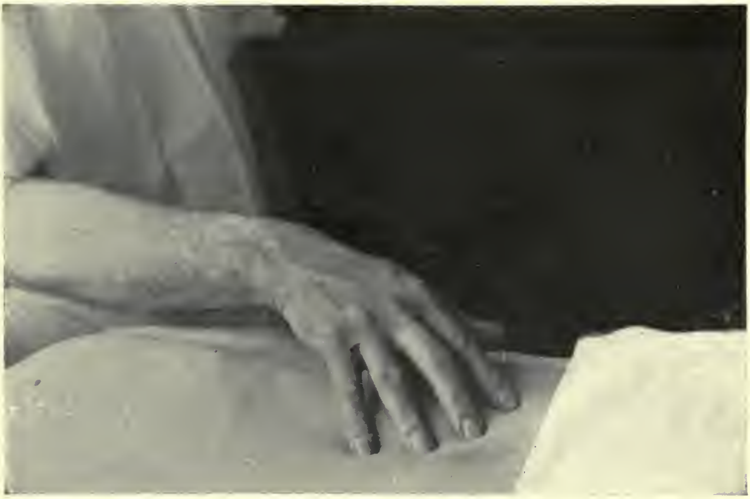

Fig. 100.—"Piano percussion."

upon another mechanical method of squeezing the gall-bladder, depending upon the diaphragm to tell the story. Standing directly behind the patient, as in the accompanying photograph (Fig. 98), provided the patient is well enough to assume an upright position,—if not, the examiner reaches over the recumbent patient from the head,—the right hand of the examiner curls up under the costal arch at the tip of the ninth cartilage, the patient is requested to take a deep breath, and, at the end of the expiration, the examining fingers seek the gall-bladder area and fix it from beneath. The diaphragm, descending from above with the beginning inspiration, brings down the liver and gall-bladder, and if a cholecystitis or cholelithic obstruction is present, the descent is checked suddenly with an accompanying groan from the patient, for the diaphragm, acting as the great piston of respiration, forces the distended, inflamed, or congested gall-bladder down upon the firmly fixed hand, and the result is an abrupt cessation of the inspiratory act, plainly shown by the patient, for he feels a severe pain.

Still another method used by Dr. Murphy is called by him the "piano percussion." It is used to demonstrate a small quantity of fluid or exudate in the abdominal cavity, resting on the hollow viscera, the intestine, and is based upon the peculiarly characteristic percussion-note given off when the abdominal wall over the fluid is struck by the four fingers, one after the other, beginning with the fourth and rapidly going to the index-finger (1, 2, 3, 4, as shown in Figs. 99 and 100). There is a complete absence of the tympanitic note which one gets in this condition when the ordinary percussion methods are used, for with the usual percussion stroke the tympanitic note from the distended bowel so obscures the faint flat note coming from the fluid that the latter is entirely lost. This "piano percussion" is so superficial a stroke, or rather series of strokes, falling almost simultaneously, that only the flat note from the fluid is brought out. It is a somewhat delicate stroke, and requires practice to make the fingers fall properly and in order, but it is of considerable value, and will help to prevent an error in diagnosis.

And, finally, to one other method attention is called. It is most simple, and should be used by every careful examiner: The simultaneous palpation of both iliac fossæ in cases of suspected acute appendiceal involvement. We have all noted in cases where there is considerable trouble in the appendix that the affected side offers greater resistance to the examining hand. We palpate, first, the healthy side, and then the other; and see quite a difference, especially in typical cases; but Dr. Murphy has pointed out, and proved by clinical results during the past twenty years (since 1889), that decidedly more information is gained when both sides are palpated simultaneously. Compare the way in which the examiner's left hand sinks into the patient's iliac fossa to the lessened descent of his right hand on the side of the inflamed appendix. The difference is grossly apparent in the typical cases. So it is with the lightly involved appendices—those cases with slight recurring attacks—the atypical cases—that this test is most serviceable. An equal pressure on the two sides at the same time, "comparative bimanual examination," will show a difference which, with practice, is discernible in practically all cases where there exists an acute pathologic condition within or around the appendix.

In reporting these methods it is hoped that a working knowledge and use of these procedures will prove of value in that class of difficult cases to which they are applicable, and that it may stimulate also an interest in that most essential field of work—differential diagnosis.

VOLUME I *NUMBER 4*

THE
SURGICAL CLINICS
OF
JOHN B. MURPHY, M.D.
AT
MERCY HOSPITAL, CHICAGO

AUGUST, 1912

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SURGICAL CLINICS
OF
JOHN B. MURPHY
Volume I Number 4

ACUTE APPENDICITIS AND PNEUMONIA

HISTORY

This child is seven years old. He entered Mercy Hospital March 9, 1912, because of abdominal pain and tenderness. Family history is negative as to appendicitis and tuberculosis. Aside from the present trouble, he has been a very strong, rugged boy.

Present Trouble.—The patient's father states that his boy's trouble dates from the time when he was one year old. Then, and on about four or five other occasions before he was five years old, he had attacks of sickness about which the father remembers little, except that he had pain in the abdomen and vomited. He is fairly certain that his boy had fever during some of these attacks, but does not know about tenderness or chills. He was sick three or four days at each of these attacks, and between them he had no pain. For three or four years he complained of epigastric oppression and the eructation of gas. During that time he has been constipated, always worse during the attacks.

In March, 1911, patient had pain in the lower right quadrant of the abdomen. It was severe, colicky, and intermittent in nature. It was present all over the central portion of abdomen, but most severe in the region of McBurney's point. A doctor was called, and finding temperature and tenderness at McBurney's point in addition to the above-named symptoms, diagnosed appendicitis and advised operative treatment. After five days in bed the child apparently was well again.

September 28, 1911, he was again seized with pain in the right iliac region. It was more severe than it had ever been. He vomited and had tenderness over the abdomen, but was all right in three days.

During the first days of January, 1912, he had another attack of pain and emesis keeping him sick for one day.

On the morning of March 6th, three days ago, his present attack began with severe pain generalized over the abdomen, but in a very few minutes becoming more pronounced at McBurney's point. Two hours later he vomited. Pain and emesis recurred intermittently all that day and until 3 o'clock of the morning following, when they subsided somewhat after the administration of an anodyne by a doctor. Tenderness in the right iliac region has been acute since the onset of this attack and still continues so.

On entering the hospital, March 7, 1912, temperature was 101° F.; pulse, 136. Two hours later the pulse was 160 and the temperature 102° F.

Leukocyte count, 16,800. Urine negative.

Physical Findings.—Examination of heart negative. Examination of chest revealed increased breath-sounds, bronchial breathing, and loud râles all over front of chest, but much worse on the right side.

Operation March 15, 1912.

COMMENTS AND OPERATION

DR. MURPHY: Please follow the history of this case carefully, as it varies from the regular course.

(Intern reads history.)

DR. MURPHY: What do you mean by "during the month of March"?

THE INTERN: Doctor, he knew it was March, 1911, but he could not tell me when it was.

DR. MURPHY: Give a distinct time in the month. "During" implies a space. If you say "in the month of March," it would mean at some time in the month of March, but "during the month of March" means he had it all through the month.

(The intern reads more of the history.)

DR. MURPHY: This boy came into the hospital on the ninth of this month, and we saw him on the evening of the ninth, when he had a temperature of 102° F.; he had bronchial râles, short and painful breathing, and we diagnosed pneumonia with acute appendicitis. He was very ill with his pneumonia. His breathing was rapid and short. His abdomen was distended. He was very sensitive in his right iliac fossa, but the operation for appendicitis in an acute stage of pneumonia results very unfavorably in the large percentage of the cases and is not justifiable.

We felt we had better let him get over his pneumonia and get a subsidence of his appendicitis, until he has reached the stage where we could operate with comparative safety. His pneumonia cleared up, and his temperature went down to about normal, but is now going up again. He has an area of sensitiveness and resistance which I interpret as a mass in his right iliac fossa. The temperature yesterday was 103° F.; that means he has a secondary infection of a new zone around his appendiceal abscess. It has been present during the course of his pneumonia, and he is in danger of having an acute thrombophlebitis of the portal veins and a rupture of the pus into the peritoneal cavity.

On this occasion we will make no effort at all to take out his appendix unless it has sloughed and is loose. We will endeavor to conduct the pus from that abscess to the nearest point of outlet, with the least possible soiling of his peritoneum, and nothing more. When he is finally over his attack, we will take out his appendix, for in all probability he is going to have other attacks. I remember a case of acute appendicitis we had ten days or two weeks ago, in which the appendix had sloughed entirely free and came out with the pus in two gangrenous masses.

Relative to pneumonia and pain in the right side of the abdomen, even down to the right iliac region, that is an important condition, as we have so many patients coming in to be operated on for appendicitis associated with diaphragmatic or pleuritic pains. They have pain in and fixation of the abdomen, and pronounced elevation of temperature. I recall an early experience of that kind when I first began to take out appendices. I was called 100 miles from Chicago by telephone to come immediately to see a case of intestinal obstruction. The doctor said over the phone that the patient had been sick about three days; that the abdomen was enormously distended; that he had some vomiting in the early stage; there were no bowel movements from the beginning, and it was thought he had intestinal obstruction. The doctor informed me that he had a temperature of 103° F. the first day. It can be laid down as a law that in the first days of mechanical obstruction of the intestine these patients never have an elevation of temperature, but late in the mechanical obstruction patients may have elevation of temperature associated with gangrene and infection. A hypodermic injection of morphin stopped the pain from which the patient was suffering. I concluded that the patient had an acute appendicitis and made the trip. On examination I found the abdomen enormously distended; the breathing much accelerated and labored, the pulse 160, and the lips cyanotic. There was a complete consolidation of the lower lobe of the right lung. There was no particular sensitiveness over the McBurney point nor in any portion of the abdomen. Friction sounds were heard all over the lower right lobe. This was a straight case of pneumonia with diaphragmatic and costal pleurisy, and the absence of bowel movements was due to the reflex paralysis of the bowel so commonly associated with this disease, and so frequently diagnosed as an abdominal lesion, such as appendicitis, cholecystitis, intestinal obstruction, peritonitis, etc. There was a reflex spasm of the intestine, and the patient had a bowel movement! Professor James Adams Allen, one of my best teachers, used to say: "What will you do when you have obstruction of the bowel and can't get a bowel movement? Give a cathartic?—yes and no. Give strychnin to stimulate peristalsis; give morphin?—yes and no. Why give morphin when you cannot get a bowel movement? Does not that stop peristalsis? Yes, but when peristalsis has ceased from reflex causes, as the passage of a ureteral calculus, hepatic calculus, diaphragmatic pleurisy, strangulation of the ovary, etc., morphin stops the reflex pain, and permits peristalsis to come on and produces a bowel movement." We will now open the abdomen over the mass in the right iliac region in the usual manner. The abscess is high up and behind the great mesentery. It formed in a very unusual position, behind the coils of the intestine, and down under the mesentery. This accounts for the great danger of pylephlebitis. Here is a place where you have to handle conditions with the greatest of care. The abscess burrowed posteriorly and behind the coils of the intestine. It is situated on the vena cava and aorta. This type of mesenteric appendiceal abscess is described in my article on Appendicitis in Keen's Surgery.

Let the records show that there were agglutinations of the bowel; the abdomen was free from adhesions in front; the base of the appendix was exposed by lifting the caput coli; the abscess was not in that location; it was found that the appendix had curved inward and the abscess had formed behind the coils of the intestine toward the midline—the umbilical zone. Here you can see the great mesentery covering the abscess as it is exposed. I will open it now with blunt dissection through the mesentery, so as not to injure the mesenteric arteries and veins, as they bleed profusely. A tube is now inserted into the cavity, and from it a large quantity of pus drains freely, and continues to drain, during the completion of the operation, which was accomplished in the usual manner. I put in a few strands of gauze around this tube, and it is the first time that I leave gauze in an abdomen after an operation for appendiceal abscess in over two years. [The patient made an uneventful recovery.]

CHRONIC APPENDICITIS

HISTORY

FEMALE, aged nineteen, single, by occupation a printer. Family history negative. Had ordinary diseases of childhood.

Began to menstruate at fourteen, always irregular, each period lasting seven days; flow moderate, three or four napkins a day. Some pain on first day of each menstruation. Two months ago had slight vaginal discharge. Occasional pain on urination. Last period Saturday, October 7, 1911. Menstruating at present. Obstetrical and venereal history negative.

Present Trouble.—September 21, 1911, patient was seized with pain in epigastrium, which became general in about two hours, and in about five hours localized at McBurney's point. Nausea began almost simultaneously with pain, and in about two hours she began to vomit a greenish-yellow material; the emesis lasted all night. She also felt very feverish during the night. The following day she felt better, but was very tender at McBurney's point. The second day she got out of bed, and on the third day returned to her work. Since this time her left side has felt weak.

At about 9 P. M. on Tuesday, October 3, 1911, patient began to have general epigastric pains, which rapidly increased in severity. Toward morning she felt very much nauseated, and next forenoon vomited once. On Wednesday pain was localized at McBurney's point, where it has remained. She vomited again on Thursday, and had a temperature of 102° to 103° F. She has not vomited since Thursday, October 5th, but has had constant pain at McBurney's point; she is also very tender. Has felt feverish since onset. No lumbar pains and no urinary symptoms. No history of gall-bladder infection, and no tenderness over gall-bladder. No history of pelvic infection. Bowels very constipated.

October 9, 1911: On opening the peritoneum intestines appeared highly inflamed and thickened, and there was evidence of localized peritonitis. In attempting to expose appendix the pus, which had been walled off in right lower quadrant, escaped in large quantities and was of a very foul odor. No attempt was made to remove the appendix, but a large drainage-tube was inserted into the right iliac fossa.

Abdomen closed in usual way. Recovery uneventful.

The patient left the hospital on November 27, 1911, and the incision closed up entirely and ceased to discharge on December 4, 1911. It has not reopened or discharged any pus since that time. She had no pain or symptoms of any kind following the closure of the wound until Thursday, December 21, 1911. The patient states that she had retired for the night and was awakened at 2 A. M. by severe generalized abdominal pains. A few minutes later she was nauseated and vomited a greenish fluid. This was followed immediately by a chill and she felt feverish. She vomited again on Friday, the day following. She has had generalized dull abdominal pains since then, up to the present time, and the abdomen is tender, especially on the right side. Her temperature was 102.6° F. on December 25th. Her temperature on admission to the hospital, December 26th, was 100° F., and the pulse 110.

Examination shows an area of dulness extending from one inch below the incision on the right side down into the flanks on the same side. The superior and inferior margins of dulness reach from the superior and inferior extremities of the cicatrix from the previous incision.

Operation December 26, 1911: Incision was made parallel with the old cicatrix. The peritoneum was much thickened, with many adhesions. Pus was present. A tube was inserted into the walled-off peritoneal cavity—the site of the pus. Another flat drain was placed extraperitoneally, to be removed in forty-eight hours. She had been previously operated for appendicitis, but the appendix was not removed at the time—it was only drained.

February 7, 1912; Patient returned for removal of appendix.

COMMENTS AND OPERATION

The history of this case is instructive in this fact: she had an acute attack of appendicitis on the twenty-first day of September, 1911. She entered the hospital with a large abscess in the right side, and was operated on October 9th, about eighteen days after the onset of the symptoms. She was in bad condition, with a very large and offensive abscess.

No effort is made by us to remove the appendix in these cases under these circumstances unless it comes right into the field, unless it is easily accessible. We do not tear up the adhesions; we do not spread the pus into other areas of the abdomen. It is an entirely different situation where an appendix case comes to us on the second, third, or fourth day. There we have adhesions of the inorganic type—natural agglutinations; while on the eighteenth day we have organic adhesions. When we separate the agglutinations we separate two non-absorbing surfaces. The lymph-spaces are coffer-dammed, and there is not the slightest danger of absorption from the surfaces. When we separate organic adhesions, then we tear up vascular areas—we produce a fresh vaccination zone, and we have all the dangers of infection which we would have if the patient had had no previous inflammatory condition. You see, then, the reason for taking out the appendix on the second or third day in the presence of pus, and also the reason for not taking it out on the twelfth or fifteenth day, because in the first case you only separate agglutinations without courting infection, and in the other you tear up organic adhesions which are easily infected.

The second operation, which was done by Dr. Golden, was a retroperitoneal operation. The abscess was opened and drained without any attempt being made to tear up the adhesions or to find the appendix. She was informed that if she had a second attack of appendicitis she was to come back on the first day of the attack, and we would be able to take out the appendix in the first twenty-four hours. She failed to follow our instructions. She did not come back the second time until she had a large abscess. If we felt she was not going to follow instructions when here the first time, we would have kept her here, as we have now, and removed her appendix.

We went into this case with the understanding that there are adhesions and the usual difficulties which one expects to encounter, and we are going to try and meet them, at the same time removing the appendix and saving her life. You will remember there was a time in the management of cases of appendicitis when we believed that the right technic was to go in and tear up the adhesions and get out the appendix and make what was known as a one-sitting complete operation. This complete one-sitting operation in 1902 received the greatest indorsement before the meeting of the American Medical Association at Saratoga. At that meeting it was shown that the men who followed the plan of doing a one-sitting operation, or tearing up everything in sight until everything was all cleared, until all possible accumulations were removed and the appendix taken out, had a mortality which was equal to, if not greater than, the mortality of the unoperated cases. At that meeting it was definitely determined that such a line of procedure was not the proper one.

There are adhesions here where the drain was put in the first operation. They are rather firm. Notwithstanding the extensive inflammatory conditions that were present here at the time of the previous operation, absorption has taken place in every position except where there was an organic fixation to the abdominal wall through the drainage-tube, and this is the position of organic fixation to the abdominal wall. It was in the line of the drain. I am freeing that now so as to endeavor to come down into the zone of the appendix, if the appendix is still here and has not sloughed off in either of the attacks. I follow the caput coli to see what is left of the appendix. There is apparently the appendiceal stump. The line of thickening at this point is covered with adhesions. I am freeing them and endeavoring to observe the location of the appendix. There is the stump. Let us see how much of an appendix is left. Here is an appendix, I think. It is embedded or apparently separated from the intestine. It is either the appendix or a tumor. The reason I think it is the appendix is on account of the mesentery, but it is separated from its organic connection. I will follow that down in order to determine what it is. This girl never could be well because she has occlusion of the appendix just distal to the caput coli. She has a chronic continued infection all the time. She had an obliterating appendicitis, which was so beautifully described by Senn. It is a very pretty picture, showing total obliteration or closure of the appendix where the acute inflammatory attack took place. We treat the mesentery of the appendix in the regular way, just as we would in an intermediate case, or in an acute case, when we are able to bring the appendix up into the field. I am putting in an overstitch to make one continuous circle of peritoneum around that, and with that stitch I ligate the mesentery of the appendix and at the same time cover the whole zone with peritoneum. You can see a chronic exudate around this appendix in the distal portion. While there is some thickening at this point, it is not very marked. That appendix would continue to fill and rupture indefinitely unless taken out. There is no drainage for the infective material in the distal portion of the appendix, and that is why it is so thickened and why it is so distended. Ninety-five per cent. phenol is applied to the stump. A puckering string suture is put in, and the stump is embedded. The first puckering string acts as a Lembert suture; the second is a Lembert suture, but not put in on the puckering string plan.

There has been a chronic inflammatory condition here, with a cobweb exudate that does not adhere and is non-infective. That is the type that we find present where there is a tuberculosis, and we all know that where there is a tuberculosis in the intestine, with this type of exudate, we are in great danger of failing to secure union. There are three means of preventing leakage there: First, the ligature around the appendix; second, the puckering string, which is a Lembert suture, and third, the accurately applied Lembert suture. I am cutting through the scar tissue that resulted from the previous operation through the line of drainage to expose the rectus muscle on the inner side. There is the belly of the rectus muscle. We do this so as to get all the support we possibly can for this line of suture.

We are operating in this case as we would on a ventral hernia. This patient has to earn a livelihood, and we must guard against a hernia at this stage by doing a thorough and systematic operation, and for that reason we are going to make an overlapping flap of the aponeurosis. The aponeurosis is on the outer side of the rectus muscle. The method of closing this incision is different from the ordinary method of closing the abdomen. We are going to make a flap closure, an ideal method for the cure of hernia,—the imbrication method, as used by Andrews in his radical operation for hernia,—and the overlapping flap method as it is used on the same principle by Mayo in his operation for umbilical hernia. The central flap is made the under flap on this occasion. The peritoneum is closed where the flap is. This is the overlapping flap, and it was put in with a continuous stitch. This type of aponeurotic fixation gives the greatest possible security that one can have against a hernia, or we might say it is the best method of preventing a hernia, and it gives the greatest security in the repair of a hernia.

Let us analyze this case from the beginning. Let us see what conclusions we can draw. First, she had the typical classic symptoms of an acute attack of appendicitis; pain, nausea and vomiting, elevation of temperature, local sensitiveness, and the formation of a large phlegmonous abscess. Second, the attack was permitted to continue until the eighteenth day before an operation was performed. As a result of that delay a large phlegmonous abscess formed within the abdomen. On opening the abdomen the condition was such that to remove the appendix at that time would materially have increased the hazard to the patient's life. The first rule of surgical practice is that a patient's life must not be hazarded unnecessarily in any procedure. The matter of whether you do a secondary operation or not is of little value to that patient or to you as compared with increasing the hazard to the patient's life. That is one thing that cannot be restored, and if you take risks, you are certain to pay the price. In these cases we learned very early, when they came to us at the stage we had here, not to do the complete operation. We have never varied from that rule, and we believe that that has been one of the sources of our success in the management of cases of appendicitis. This woman ran a great risk, and she has paid the price for disobeying our instructions, and for which we are not responsible. When she came the second time the abscess was opened extraperitoneally—the peritoneum was not opened at all. This time we did not let her go home; we kept her here in the hospital while she was convalescing from her attack. She was operated on the twenty-sixth of December, and you can see an illustration of the great absorption of adhesions that can take place in a comparatively short period of time when they are not organic. When they are organic, the absorption does not take place. That means absorption of the adhesions that formed at the entrance of the tube did not take place and never would take place. That is what is known as an organic adhesion, because the peritoneum is destroyed completely through its depth and is not covered again with endothelial cells.

Let the record show that the adhesions had practically all absorbed down to the cobweb formation on the general surface of the peritoneum; that the only organic adhesions remaining were those at the point of entrance of the drainage-tube and down the side of the parietal peritoneum, where the tube drained the depth of the abscess; that the appendix was folded between the caput coli and the ileum; that it was obliterated at its base, and that this obliteration produced the continued retention of pus in the appendix, caused its distention to almost the size of my little finger, and kept the patient in a constant condition of mild sepsis.

A Physician: Will you please tell us what suture material you are using in your figure-of-8 stitch?

Dr. Murphy: This is silkworm-gut dyed black, because it is easier to recognize when we come to remove it. We have used no other material for a great many years, and we rather like it. I do not depend alone for support on my catgut suture. This is an inabsorbable suture of two rows, because the lower loop represents one row, and the outer loop the other, and still neither one is buried—all inabsorbable material.

There was no evidence at any place in the abdomen to show that the abscess had ruptured into the intestine. If the abscess had ruptured from the appendix into the bowel, we would find organic fixed adhesions between the appendix and the bowel. There was at no time any communication between the intestine and the peri-appendicular abscess.

Let us go back to the consideration of the hazards this patient has had from the fact that her appendix was not removed early in the first attack. We can only repeat now, what we said in 1889, when we wrote our first article on lesions of the appendix, namely, that the time for operation was now! and "now" meant within the first few hours of the attack, and we repeat again the statement we made at Budapest, at the meeting of the International Medical Congress, 1909, that every case of appendicitis that is operated in which pus is found outside of the appendix at the time of operation has, in the presence of that pus, irrefutable evidence that the case was badly managed up to that time. There is no longer any reason for having pus outside of the appendix, as all cases of appendicitis should be operated promptly. The symptoms are so uniform in the acute infection, and the chronic infections never cause abscess to form outside of the appendix; the symptoms are so uniform and regular that "he who runs may read" the diagnosis, and, having read the diagnosis, he has no way of justifying himself for waiting any longer. There was a time when it was justifiable to wait. Now it is not justifiable to wait, because it is the consensus of opinion of those who have had the greatest experience, that you cannot tell, from the course of the symptoms in the first twenty-four hours, what the course is going to be in the second or third twenty-four hours. Inasmuch as you cannot tell what the course is going to be, you are not justified in permitting a case to go into this advanced stage before something radical is done.

In the recognition of appendicitis in the initial stage of the acute attack we always have a regular course. First, pain—*and pain is always first.* I was in consultation in a case this morning in which the question was raised whether or not the last attack the boy had was one of appendicitis. His trouble began with headache. He had nausea and vomiting with his headache before he had any pain in his abdomen. The pain of appendicitis is the primary symptom. The reason the pain is the primary symptom is: The appendix is made up of three coats. In all but 1 per cent. of the cases infection takes place in the mucosa; at first the mucosa swells and cannot expand on account of the firm inelastic fibrous coat. That causes the pain; that causes the colic, and then, as the mucosa cannot expand, you have exactly the same condition as you would have if a foreign body were obstructing a canal, causing reflex vomiting. There are only one or two efforts at emesis in the primary stage, and rarely ever is there vomiting in the second stage of the disease. Following that is local sensitiveness, which is due to the great distention of the appendix. Following that comes absorption, in a case of infection causing elevation of temperature and causing an increase in the number of leukocytes.

We have had some interesting observations on the relation of temperature to leukocytosis. Occasionally we have a temperature of only 99.6° to 99.8° or 100° F., with a leukocytosis of 15,000, 18,000, and 20,000. In this class of cases, when there is any question at all as to the diagnosis on account of the temperature being 99.6° to 100° F., there is a high leukocytosis, which shows that infection is present. In that way leukocytosis is of value as a diagnostic symptom in probably from 3 to 5 per cent. of the cases; otherwise you can make the diagnosis in the acute attack without a leukocyte count. You base the diagnosis on the symptoms occurring in this order: Nausea and vomiting never precede pain in appendicitis; temperature never precedes pain in appendicitis. *It is pain, nausea and vomiting, local sensitiveness, elevation of temperature, and an increase in the number of leukocytes.* That is the uniform order in which the symptoms occur.

We have had, I think, nine cases of typhoid that came to us with a diagnosis of appendicitis. In the first case we did not know whether the patient had a temperature or not before pain had developed. We operated in this case and found a typhoid ulcer in the appendix. In the second case there was elevation of temperature before the pain for three days. The typhoid ulcer demonstrated in the appendix accounted for the sudden pain, nausea, vomiting, and local sensitiveness in the third day of the fever. It usually occurs between the third and fourth day. In the third case we had we did not know whether elevation of temperature had been present before the pain, and we operated and found a typhoid ulcer in the appendix. In the remaining six cases we had all that information, and in each case we made a diagnosis of typhoid ulcer in the appendix, did not operate, and each case went through the regular course of typhoid.

You see, therefore, why I am so particular about emphasizing the fact that the temperature does not precede the pain in the ordinary case of appendicitis. It is unnecessary to operate for typhoid appendicitis, because only ½ of 1 per cent. of the cases have a perforation of the ulcer in the appendix—a much smaller percentage than the perforations which take place in the ileum and large intestine. The appendix is not prone to perforate in typhoid ulcer, while the ileum and the large intestine, even down to the sigmoid, are prone to perforate. But you do not need to operate for it, and we do not operate for it.

ANKYLOSIS OF THE KNEE; ARTHROPLASTY; JOINT INFECTIONS

HISTORY

Housewife, aged twenty-seven. Entered hospital because of ankylosis of left knee-joint. Mother died of tuberculosis of the lungs while patient was a very young child. She nursed and always lived at home with her mother.

Present Trouble.—Patient lives in Denver, Colorado, and just after Thanksgiving, 1907, she left Colorado, where it was warm and dry, and came to Illinois, where the weather was then very cold and stormy.

December 8, 1907, she was taken ill with a severe attack of tonsillitis, which kept her in bed, with fever, for one week. The day after Christmas, 1907, the patient visited friends all day, making numerous calls, walking far in the snow, getting her feet wet and becoming very tired.

At 2 o'clock in the afternoon, December 27, 1907, the patient experienced a sudden severe pain in the first phalangeal joint of the third finger of the left hand. This joint rapidly became hot, painful, swollen, and tender. One half-hour later she had a severe chill lasting fifteen minutes. She is certain that pain preceded the chill. About 9 o'clock the same night the right shoulder likewise became hot, painful, and tender, and in succession the right ankle, the right wrist, and, last, the left knee, became involved, all within the next twenty-four hours. She was kept in bed for three weeks and had much pain and a high temperature.

All joints became normal rapidly, with the exception of the left knee, which continued to be sore, painful, and swollen. She remembers that the skin over the joint pitted on pressure made by the doctor's fingers. After five weeks the swelling and tenderness gradually subsided, but the leg could not be flexed and has remained so ever since.

In June, 1908, leg was flexed under anesthesia, but stiffness still persisted. In July, 1908, another attempt was made to break up the adhesions, and a nine-pound extension was applied, but without results. An opening into joint was never made. It has never been injected.

Urine contains a trace of albumin. Leukocyte count, 8000.

COMMENTS AND OPERATION

This is a typical history in every way. The primary infection took place in her pharynx. The pharyngeal infection was of about two and a half weeks' duration, and then, as the result of additional exposure, there followed a metastatic infection in her joints. She had a chill, followed first by involvement of a number of joints, without any destruction of tissue, and finally concentration of the destructive effect in only one joint, the infection in all the other joints subsiding. That is the rule. That is the typical course of all these cases of arthritis that are called "rheumatism." They are purely cases of metastatic infection, and in the great majority of cases the primary infection can be located elsewhere in the body, most frequently in the mouth, as in this case.

Before we began to study these infections from the standpoint of metastasis, no record was made of the cold, or the sore throat, or the tonsillitis, or the abscessed tooth, dysentery, gastro-intestinal fermentation, etc., that preceded the attacks of arthritis or rheumatism two or three weeks. Why? Because we had failed to connect the primary infection with the secondary arthritis, and the lapse of time between the subsidence of the primary infection and the appearance of the secondary manifestation is usually long in the average infection, except of the streptococcus type, and it led us to overlook the fact that arthritis is a metastatic infection, easily recognizable and easily demonstrable, following a primary infection of some mucous membrane, as a rule. The pharynx is the most common seat of the primary infection, and in the recent literature you will find that authors have emphasized the importance of disease of the tonsil as a primary focus of infection. The tonsil, in our experience, has been the least common, the pharynx the most common, source of the metastatic infection. Next in order follow the nasopharynx, the accessory nasal sinuses, the teeth and gums; infections of the bronchial mucosa, infections of the lower part of the intestinal tract, such as diarrheas, gall-bladder infection, and so on. It is rather surprising that the profession failed for so many years to connect the secondary infection in the joints with the primary infection of the mucous membranes, when for many years we have recognized the relation between the primary urethral infection and the secondary articular infections of the Neisserian type. The reason we always connected this was that the patient did not get over the urethral discharge before his joints became involved, and he had a painful and sweeping reminder of its presence. The secondary metastatic infection of the joint or joints of the Neisserian type does not take place, as a rule, until the eighteenth day, and about 90 per cent. of the cases occur between the eighteenth and twenty-fourth day after the primary urethral infection. That is not an accident nor a coincidence. It is a habit, a pathologic habit, and it is in consonance with the other types of metastatic infections that occur in joints. An exception to this is the streptococcus infection, where, within forty-eight hours after the appearance of the primary invasion, there occurs the secondary metastatic manifestation in the joint or joints.

I do not think I can give you a better illustration of this than by mentioning a case that occurred in the practice of my former colleague, Dr. Neff, now of Spokane. A patient, after paying his devotions to the goddess of Cloacina, without washing his hands struck a match on the seat of his trousers, and a small sliver from the match penetrated his finger. That evening he had a chill; the next morning he had a phlegmon of his chest; no suppuration, but considerable sensitiveness. In the evening he had another chill, and the next morning the left knee was swollen and distended. He was in charge of Dr. Neff, whose ability cannot be questioned. Dr. Neff immediately incised the phlegmon on the chest, and made a culture of its contents. He withdrew blood from the arteries, made a culture, and he likewise made a culture of the aspirated fluid from the joint, and all three cultures and smears showed streptococci and colon bacilli. You see where he got the colon bacilli. Inside of forty-eight hours after the onset of the symptoms the joint was filled with fluid containing both microörganisms. It was opened without tube drainage. A number of other joints were similarly opened. He had phlegmonous abscesses elsewhere; all were opened without tube drainage. It is a law, in connection with the infections of joints, never to put in a drain. Open the joint and relieve the tension, but keep out the drain. The man in this case got perfectly well without ankylosis of a single joint. After the aspiration of the joint Dr. Neff put on Buck's extension immediately. Aspiration, no drain, Buck's extension!

It is impossible for me today to go into the details of the method of destruction of joints, but let me point out a few important mile-stones in connection with this subject.

An infected joint is destroyed primarily through the accumulation and the pressure of the products of infection held under tension in the joint, as well as the virulence of the infecting microorganism. Relieve the tension, and nature does the rest. If you have a phlegmon in the thigh, reduce the tension, and the patient experiences relief from pain. The joint has a velvety inner lining of synovial membrane. The outer lining is an inelastic, non-absorbing capsule, capable of sustaining a pressure of from 50 to 120 pounds. It is this capsule that holds the products of the infection in the joint under such colossal pressure. It is a leather bottle, so to speak, of fibrous capsule, and the products of infection cannot break out because of the non-vascularity of the capsule. Finally, the endothelial cells lining the synovial membrane are destroyed, and what results? Ankylosis. The second element in producing ankylosis is the pressing together of the bones of the joint by the contraction of the muscles above and below the joint, which occurs as the result of an endeavor to immobilize the joint. It is that involuntary muscular contraction which most frequently causes ankylosis, except in the drainage cases. In a large number of these cases we have found that there is one spot of ankylosis, as on each condyle of the knee, for example, or only one spot of ankylosis on one condyle, these being the only points of contact produced by this involuntary muscular contraction.

How can we stop the destructive effect of these two factors in the production of ankylosis? One, the increased interarticular pressure caused by the accumulated products of the infection, and the other, the muscular contraction. The latter is easily stopped in all large joints by the application of Buck's extension. All that needs to be done is to relieve the pain, and that can be effected by applying a Buck's extension. It is much better than to give 1/3 or 1/2 grain of morphin. The patient will not allow you to take off the weight, because the moment you do so the joint becomes painful. The pain is also an indication of the destruction that is being produced in the joint by pressure. Remove the pressure, and you relieve the pain and lessen the amount of destruction.

You can stop the destructive action of the accumulated products of infection in the joint in two ways—either by incision into the capsule and permitting the pus or the purulent fluid to flow out, or by aspiration and injection. By aspirating you remove all the fluid that will come out, and then inject into the joint a fluid that will render the contents of the joint sterile, or make the fluid in the joint a bad culture-medium for every type of microörganism that is likely to get into the joint.

The fluid we inject into the joint is a 2 per cent. solution of formalin in glycerin. Inject into the joint 10 or 15 c.c. of this solution that is mixed at least twenty-four hours before it is used, because it requires twenty-four hours for the formalin to dissolve with the glycerin. That renders the joint a bad culture-ground. Reaspirate the joint from day to day, or every second day, keeping the tension off, and in a little while the synovial membrane becomes coffer-dammed and absorption of septic products ceases. The formalin and glycerin solution does more than to render the joint fluid sterile, or to act as an antiseptic. It causes an enormous outpouring of the polymorphonuclear or polynuclear leukocytes. They are the real phagocytes. They have not only the power of grasping and holding the microörganisms, but they contain trypsin. That trypsin is freed on their death. It is a ferment. It converts albuminoids into peptones. This phagocytosis and digestion rapidly render the joint sterile and thus overcome the infection. Prevent ankylosis by extension, and inhibit destruction of tissue by aspiration.

Every acute infection of a joint, call it "rheumatism," arthritis, or whatever you wish, should be treated as you would treat an infection in any other tissue in the body, with the one exception—that you cannot put in a drain and allow the air to come in contact with the synovial membrane. If you do, you violate the first law to be observed in connection with synovial or serous membranes in every position in the body, namely, that exposing them to the air for a considerable length of time will cause destruction of the endothelial cells and lead to the formation of adhesions and ankylosis. These are all simple things, but every one is vital to the final result, and every one can be utilized to avoid the awful catastrophes that occur to joints as a result of infection.

This patient is one of the best cases to illustrate this. She has little or no deformity. She has practically a straight limb. The skiagram shows that there are only two points of ankylosis absolutely fixing the joint. This accentuates what I said, namely, that if she had had Buck's extension for the first two weeks of joint inflammation, she would not have had a bony ankylosis at all.

There is fixation of the patella. I do not think that it is a bony ankylosis—merely a rather firm fibrous fixation. I hope it is not bony, as it will save me turning the patella turtle. The worst trouble we have had in any of these cases has been a necrosis of the external skin-flaps, and it has been the most annoying trouble we have had. We will now make a U-shaped incision to expose the joint, commencing two inches above and one inch to the outer side of the upper end of the patella, passing downward to about the level of the tibial tubercle, and then across the tibia toward the inner side, with the convexity of the incision downward, passing upward to the inner side of the patella to a point opposite the starting-point. This incision extends through the skin and fascia down to the capsule of the joint. Next I will divide the capsule lateral to the patella to get my interposing flaps, which have to go in between the joint surfaces subsequently. One flap will cover the outer condyle, the other will cover the inner condyle. A U-shaped incision is made on both sides of the knee, the base of the flap being directed downward and on a level with the articular surface of the tibia. The next thing to do is to loosen this patella. There is only a fibrous fixation. That is a great relief.

In this case I will not need the long tissue flap, nor will I need to rotate the patella. Next, we will find the line of demarcation between the femur and the tibia. I am following that line around so as not to disturb it any more than is necessary. I shall free this patella from its fibrous attachment to the femur.

Now we are in the joint zone. The posterior portion of the capsule has not been touched. It is intact. The lateral portions have been converted into the interposing flaps. We are ready to make the new bone conformation for the joint. This conformation of the ends of the bones is made with straight and curved chisels of various sizes. Sufficient allowance must be made for the interposing flaps, and the leg must be plumb. We keep the intercondyloid ridge on the tibia intact. That is essential to the final result. If you do not keep this elevation intact, you will have a slipping of the femur from side to side. You see how much space I have here between the femur and tibia—$\frac{1}{3}$ of an inch. We are getting ready now to put in the interposing flaps. The flap which was taken off the inner side I will now suture to the stump of the crucial ligament behind, so that it covers all the articular surfaces of the internal condyle of the femur and the inner tuberosity of the tibia. For suture material we use phosphor-bronze wire, 12 strand, not chromicized gut. It is non-absorbable, flexible, and is not irritating. The outer flap will cover the external condyle. It is drawn in at a different plane, because it goes back to the internal condyle. It overlaps the edge of the internal flap. I will fix this in position so as to cover the margin of the tibia and catch the periosteum of the tibia. I am putting in just one under-stitch, so as to be sure and have this flap overlap in the middle, because there is where you have the greatest danger now.

We are endeavoring to do here just what nature does in causing non-union of a fracture—interposing fascia and fat between the bone-ends. We know that in ununited fractures this is the cause of most cases of failure of union. Here, that is just what we want.

The next step is to place a portion of this flap which I have brought from the vastus internus between the femur and the patella to prevent the recurrence of fibrous adhesions between these two bones. We could not get bony union because we do not have a cartilaginous surface, but we would get a fibrous fixation which is equivalent to bony union as far as the result is concerned. The remnant of the vastus internus I am fixing to the patella, so that it will not subluxate outward. I have to turn the patella bottom side up, and in doing that we have the disadvantage of traction. So much for the upper stability of the patella.

For the lower stability of the patella, here is the tendon patellæ and there is the fascia. We bring that back and unite it, so that it cannot slip to the outer side. The subsequent luxation of the patella is always to the outer side. One rarely ever has to put on a ligature to stop bleeding. All oozing can be stopped by compression. I will secure the sides of the ligamentum patellæ, so that it will not tend to inversion or eversion subsequently, but will stay in the exact relationship to the tibia in which it was before.

This is the same plan we have followed in all our cases, with the exception of the management of the patella. We manage the patella in four different ways, depending on the case:

1. By using an interposing flap from the vastus externus and vastus internus to prevent reconsolidation of the under surface of the patella with the femur.

2. Splitting it into two parts from above downward, and then turning the upper half under the lower half, so that the smooth aponeurotic surface came next to the femur, thus making it impossible for bony union to take place.

3. Freeing the vastus externus and internus attachments for a distance of two inches above and below the patella; next dislocating it from side to side during the operation; and when the limb is straightened out and the interposing flap is in position, a 180 degree rotation of the patella is made, so that the upper surface of the patella now becomes its articular surface. The vastus externus and internus are now sutured to the opposite sides from which they were freed, to prevent luxation of the patella and the return to its former position.

4. Covering the under surface of the patella and the entire articular surface of the femur with a fascial graft taken from and detached from the trochanteric zone of the fascia lata, without rotation of the patella.

I have had good results with all these methods. The rotation, however, is the simplest one, and after operation gives an additional leverage to the quadriceps tendon. It has some disadvantages, as it taxes the vitality of the skin-flaps.

There is a little oozing from the surface of the bone. I am taking the tension off of the line of skin suture and putting it on the deeper structures. The tendency of the flap is to pull outward. I have put that away back so as not to interfere with the circulation at the edge. We close the wound in the usual way, using catgut for the deep sutures and horsehair to approximate the skin incision. Subiodid of bismuth powder is dusted on the wound, and this is then sealed with gauze saturated with collodion. A four-fifths plaster-of-Paris cast is applied, extending from near the groin to below the ankle. There is practically no tension at all on the line of suture. Remember, I fear necrosis of this flap more than anything else in connection with this operation.

Let us recapitulate: In this case the ankylosis was of the condyles close to the middle line, near the intercondyloid ridge of the tibia, and the patella was merely fixed by fibrous adhesions, as interpreted by us before operation. Both of these suppositions were sustained by the findings at the operation. This is rather extensive work, and involves a certain hazard in connection with infection. That is the greatest danger of all these procedures, because you have to change your position often, and because you have so many instruments, sponges, etc., in the field.

We have had one recurrence of ankylosis of the fibrous type in an elbow. The patient left the hospital shortly after getting out of bed. She would not submit to the massage and exercise that were ordered after the operation. The result was that the ankylosis recurred and the arm is as stiff now as it was before the operation. We have not had a single recurrence of a bony ankylosis of the knee. We have had one infection, namely, osteomyelitis of the femur, following an operation with some necrosis. We had an infection in a case of hip ankylosis where the patient, a morphin habitué, was deprived of the morphin, and on the twelfth day after the operation he tore off the dressing from the wound; infection set in, and he died three weeks afterward from infection. It was the second operation on the same patient. We had operated previously on the other hip and secured a splendid result.

The best joints in which to do the operation are, first, the hip; second, the elbow; third, the knee. The knee requires the greatest mechanical adjustment of any of the joints. Unless the limb is perfectly plumb after the operation, the knee will be drawn outward or inward. In chiseling the ends of the femur and tibia so as to restore their normal anatomic conformation I endeavored to get it exactly plumb, so that the pressure would fall evenly on the external and internal tuberosities of the tibia and on the external and internal condyles of the femur. In this way we hope to be able to have this patient carry her weight without the slightest deviation of the plumb-line of the leg. It was not so difficult to do that in this case as it has been in many of the other cases, because here we had the limb plumb to start with. In the great bulk of our cases we have had great deformity associated with the ankylosis. In some of them we have had the angle so acute that the heel practically touched the buttocks. The angle at which the ankylosis takes place very materially increases or diminishes the risk which one assumes in doing the work. It also adds to the mechanical difficulty of the operation. The straight limbs afford the best conditions for securing good mechanical results.

After the skin wound has been carefully closed with horsehair, the site of operation is dusted freely with subiodid of bismuth. It is a desiccating powder, rather than an antiseptic. We never drain in these cases. A moist carbolic gauze dressing is then applied. The reason we use carbolic gauze dressing and not the ordinary dry gauze is that there is always some bloody oozing into the gauze, and in the dry gauze the blood decomposes. If it oozes into this carbolic gauze dressing, it does not decompose. It remains sterile; no saprophytes can live in it. It is the saprophytes which we fear. There is only the slightest pressure over the flap. There is no pressure at all over the patella. That is where the greatest danger is. We shall put on a Buck's extension with a ten-pound weight. The leg is placed in a wire cage and elevated from 14 to 16 inches, otherwise the foot will swell. We use a double-inclined plane. The heel will not be hazarded in this case.

In doing this work you are on edge all the time. Every single step must fit to get a result. It is the little things that count in all the technic of joint work. They must be done with precision.

Here is a patient who had an ankylosis of both hips. The first operation was done seven or eight months ago. The second operation was performed two weeks ago. He also had an ankylosis of his spine extending to his second cervical vertebra as the result of so-called rheumatism. Both hips were fixed. He could not sit down for nine years, because both of his hips were ankylosed. Now he has a movable joint. He can bring his leg up to a right angle with his body, swing it, abduct it and adduct it. He also has a considerable degree of rotation. Before we operated on the second leg he could bring the leg operated on first almost to or better than right angles, showing what can be done in these cases.

Here are some illustrations of another case. (See Vol. I, No. 3, p. 301.) This was a most difficult case from a mechanical standpoint. The ankylosis of the knee began when the patient was seven years of age—some thirty years ago. We encountered enormous difficulty in getting the leg straight. It was bent so much that with the thumb on the buttock and the fingers spread, the little finger touched the heel. The femur was bound. We turned the patella turtle in this case. We had to remove an inch and a half of the femur in order to get a straight leg, and still we secured a good anatomic conformation. In straightening the leg we had to stretch the nerve and blood-vessels so much that we feared gangrene would occur. She had complete paralysis of the foot from stretching of the nerve. That paralysis is rapidly disappearing. She was operated on fourteen weeks ago. There is perfect freedom of motion of the patella. We turned the patella over, so as to put the prepatellar bursa underneath to form the lining of the joint. She has a little ulcer on the heel, because she had no sensation, and the pressure on the heel caused the ulceration. The flexors are already acting; the extensors will be acting shortly.

Here is a case (see Vol. I, No. 3, p. 303) in which we did another type of operation for ankylosis. We had to take off the head of the tibia down to about the level of the fibula. The patient had been operated on before elsewhere, and there was so much cicatricial tissue around and about the knee that we could not secure the customary interposing flaps. We removed the trochanteric fascia lata and used it as the interposing flap. We have succeeded in getting splendid separation of the bone, and we are going to get an excellent result. That is the final thing I planned to do nine years ago. I began doing flap operations of one type after another. I predicted then that the time would come when I would be able to take a flap from some other portion of the body and place it between the bones to serve as the interposing flap, but I had never had a case in which I was forced to do it, and inasmuch as I was getting good results with the usual method of making these flaps, I did not feel justified to try a new method. She had no patella, the patella being merged into the femur. I chiseled that out and made a new patella. There was healing by primary union. She has some function, but not as good as it would be if she could be prevailed on to carry out the passive motion and exercises prescribed in these cases. The anatomic result is excellent.

The next case was one of very bad Pott's fracture with a posterior luxation of the astragalus. (See Vol. I, No. 3, p. 451.) The operation was done several weeks ago. We brought the astragalus a little anterior. We refractured the internal malleolus because it had united to the tibia in a position which caused the anterior displacement of the bones of the leg. Then we nailed both malleoli into their normal positions with wire nails. The result is excellent. We have bony union, and all the deformity has disappeared. The leg is straight, the foot can be placed squarely on the ground, so that she has a perfectly normal leg. Motion is already very good.

Here is another illustration of a Pott's fracture complicated by a spirochetic infection. (See Vol. I, No. 2, p. 151.) There was inversion of the foot with absorption of bone. We took out all the dead bone, freshened the surfaces, and drove in a nail from below through the astragalus and straight up into the tibia. We hope to get bony ankylosis in a Charcot joint. He has a serviceable ankle. We also drove a nail through the tibia and fibula as a lateral support and splint. We fractured the fibula above the joint. He will have a bony ankylosis in his ankle, so that he can use it and make his living.

The next case is one of a fracture through the anatomic neck of the humerus, with dislocation of the head of the bone downward into the subglenoid space. The patient came to us about three months after the accident. We liberated the head, took it out on the table, freshened the fractured surface, replaced it in the glenoid cavity, and nailed it in place with a wire nail driven in from the outer aspect of the humerus at the greater tuberosity. There is perfect regeneration of the head of the bone, with return of normal function in the joint. We secured the same result in a hip case. The fragment, consisting of the head of the femur which had been broken off from the bone at the anatomic neck, was rotated and had strangulated the ligamentum teres. The patient came in seven weeks after the accident. The head was dead but aseptic. We did not take it out; we turned it around and nailed it back in place. The head of the bone has been completely regenerated. It is now about three years after the operation. Function is normal in every respect. There is a shortening of less than half an inch.

Bone always regenerates, no matter how long a transplant you put in, if you contact it at both ends with living osteogenetic elements. The transplant acts as a support for the Haversian vessels going from the living bone into the Haversian canals of the dead bone, carrying the osteoblasts and osteoclasts and filling up all the dead portion. If you take away all the fragments of dead bone, the osteoblasts will make new bone of identically the same shape and size as the normal bone. The only rôle the transplant plays is a mechanical one, that of carrying the Haversian vessels.

This is a case of ununited fracture of the humerus. (See Vol. I, No. 2, p. 193.) A Lane plate was used to maintain perfect approximation and still there was failure of union. We took out the Lane plate, waited until healing had taken place, and then put in a 4½-inch splint taken from his tibia. The skiagram shows that bone regeneration is progressing rapidly.

We recognize five indications for transplantation: First, in ununited fractures; we never adjust ununited fractures with plates or wire. We put in an interosseous splint. Second, for the removal of infective diseases of bone, as in osteomyelitis, where there is much necrosis of a portion of bone. Take out the dead bone and allow healing to take place. Third, for the removal of non-malignant neoplasms, that is, of the fibrous and cystic variety of bone disease. Fourth, for the filling up of aplasic conditions where the bones fail to develop as they do in the arm and tibia. Fifth, for malignant diseases of bone, as sarcoma. These are the conditions in which regeneration of bone can be effected with mathematical certainty if you secure and maintain perfect asepsis. We have not had a single case of failure from sepsis; not one. In arthroplasty, if, following a transplantation of bone, sepsis should occur, you must take out the transplant, do the work over again, and do it better than you did the first time.

I demonstrate these cases so that you may note their progress from week to week, and in that way get a better idea of what is to be expected from such cases.

ANGIOPHLEBITIS OF LEG AND THIGH—OLD MUSCULAR HEMANGIOMA

HISTORY

Mr. M., clerk, aged thirty-seven, entered hospital because of a deformed foot and leg. Had malaria of the tertian type at twelve. Recovery excellent. Never had rheumatism or any soreness or disease in any other part of body. Never used alcohol in any form or any other drug. Neisserian infection eight years ago. Recovery apparently absolute. No joint or bone pains afterward. Denies lues in family or self. No tuberculosis or malignancy.

Present Trouble.—At the age of seven he noticed that he could not flex the right foot dorsally, though he could run and play without limping. He wore out the sole of the right shoe rapidly on the inner side. When ten years old, he first noticed that his right leg would not touch the floor when standing erect. Right leg was then slightly smaller in circumference, and his mother noticed that the outer side of the right calf presented a sunken appearance. The deformity seemed to develop gradually, the foot and leg drawing up more each year. Ten or twelve years ago the hamstring muscles began to contract, pulling leg upward until it cannot now be straightened out at all. The fibular region has been sunken for the past ten or fifteen years. Leg never has been injured in any way, nor has it discharged pus, nor has there been a sinus leading into it. The veins on the dorsum of the foot are distended and calcified. A very slight tenderness has existed in the limb during his whole life. Foot has never felt cold and never has been the seat of edema or swelling.

Examination by Intern.—No evidence of rickets found. No asymmetry of head or chest, but mandible is unusually weak and prominent at posterior portion near the angles. Talipes equinus, with a little varus, of right foot. Contour of the fibular region not maintained; presents sunken appearance, with thickening and bony feeling deep under skin at posterior and lower portion. Both hamstrings are contracted, and on deep palpation posterior thigh feels slightly indurated in places. Lungs and heart are apparently normal. Urine normal.

Further Examination by Dr. Murphy.—A venous angioma of right foot is present. It seems to be typical of the changes found in all these cases. Atrophy of both muscles and bones has occurred. Venous angioma exists in both gastrocnemii and in the soleus muscle, but mostly the gastrocnemii. It extends upward from five inches above attachment of the tendo Achillis. It is hard, woody, and pains on deep pressure. Posterior tibial artery is plainly visible through the skin behind the internal malleolus. It looks calcareous, but this is not confirmed by palpation. Patient was informed that operation would put excessive tension on vessels of popliteal space and might cause gangrene. The two internal hamstrings will need to be divided. Three tendoplasties will have to be performed on the inner side of the foot. Adductor group of muscles is very materially shortened.

COMMENTS AND OPERATION

We have had cases similar to this at this clinic, but not in the stage in which this man presents himself to us. The description of the veins, as it was read by the intern, is not correct. He apparently had an enlargement of the veins on the anterior surface of the foot. That is my recollection. From the time he began to walk he always wore the sole of the shoe out near his toes, and it was difficult for him to get his heel down. The key to the situation is the fact that he had an enlargement of the veins of the foot and toes from birth, and that is what led to the diagnosis. He has now no special enlargement, but still there is some enlargement of these veins, and when you palpate his toes, you will find he has a diffuse venous angioma of the lower half of the great toe. The muscles are all absorbed, the vitality

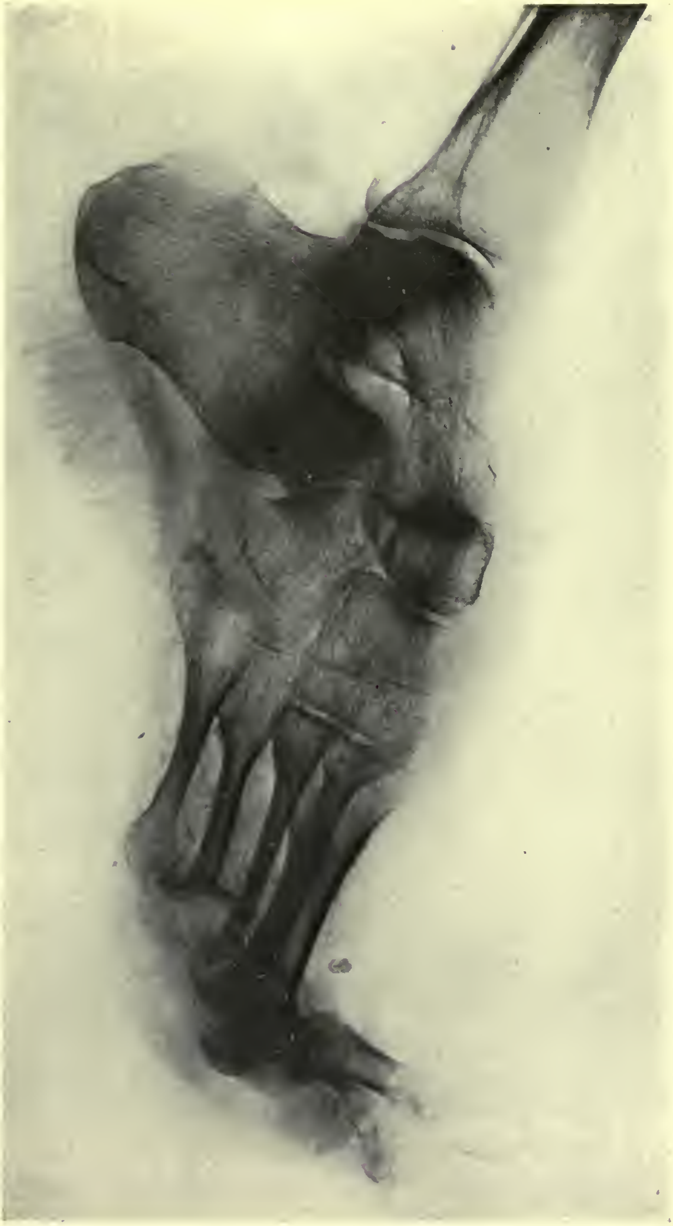

Fig. 101.—Skiagram showing calcareous deposits in small vessels of foot.

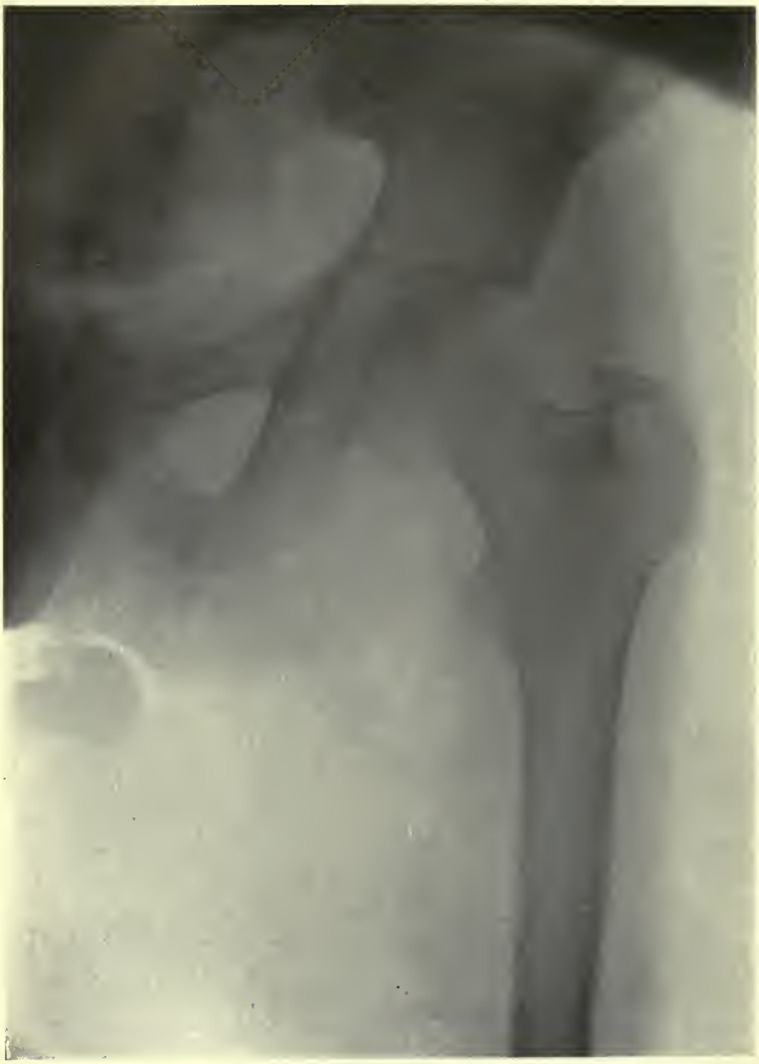

Fig. 102.—Skiagram showing vessels of upper two-thirds of thigh free from deposits.

of the tissue is greatly impaired, and it feels like bone surrounded by skin with a soft cushion of fluid beneath.

When you palpate two inches above the tubercle of the os calcis, you will find there is a solid mass running all the way up to his popliteal space, and extending through the popliteal space and up four or five inches in the fold of his thigh. It is hard, even bony in many places, and always sensitive to pressure. The talipes equinovarus—all the deformity—is due to the continued and increasing constriction of his gastrocnemius and soleus muscles.

When you feel the muscle, you ask yourselves the question, Is this a case of myositis ossificans or of some other lesion? It feels as hard as bone. The key to the situation, however, is the fact that on the foot there is still present a venous angioma. He had a venous angioma originally involving his gastrocnemius muscle, the popliteal space, and the lower third of the thigh. That venous angioma had a phlebitis start in it, and then he had a thrombophlebitis, with the formation of phleboliths, all through that muscle. Then came the gradual contraction of the muscle. The skiagram shows the condition beautifully. There is periphlebitis extending down to the external malleolus. There are two inches in which there is no deposit, commencing on the outer side, over the articulation of the fibula, then extending up from the attachment of the biceps to the fibula, then passing up and appearing again on the inner side. There is an active venous angioma, without thrombosis, in the popliteal space.

We want to try and get the foot straight. I want to see how straight the knee comes under traction. I have to let the leg move back as soon as I straighten it, because the heads of the gastrocnemius muscles are attached to the posterior outer aspect of the external and internal condyles of the femur, above the level of their articulation. Stretching that by any means except by operation would be ineffectual. When we free this, it is a question how much trouble we will have with the capsule of the joint.

This foot has been in this position since birth, I understand, so that we are likely to have trouble in lengthening the posterior capsule of the joint, and we may have to do a capsulotomy on it.

There is danger of gangrene occurring in this leg in two places, and this danger has been distinctly stated to the patient. The artery is diseased, thickened, and thrombosed, so that we have a fear of fracturing the posterior tibial artery when we bring the foot forward, and that may cause gangrene. When we make extension of the leg, we may produce sufficient compression on the popliteal artery to interfere with the circulation in the popliteal space; it may be compressed by a phlebolith, and then gangrene of the leg would occur. This man had to be prepared for the dangers of this operation, and he had to accept them. A positive statement was given him before we decided to operate. That is the proper way to deal with these cases. We cannot assume the responsibility for him. He must know the danger to which he is exposed.

The tendon of the gastrocnemius is displaced inward by the phlebolith. We are not going to cut out the phlebolith, but we will elongate the tendon. The superficial veins we must expect to be enlarged here. Why? Because the deep ones are occluded.

We will make the first incision longitudinally over the tendo Achillis, which we will elongate in order to put his foot into a normal position. We draw the foot away up and divide the tendo Achillis by a double L (˥L) shaped incision. We need an elongation of nearly three inches. Therefore we cut across the tendon half way at one point, from without inward, then half way again, but from within outward, about an inch and a half below the first incision, and unite the two transverse incisions by a longitudinal incision through the center of the tendon. You can see that is just a little over an inch and a half division. By letting these two pieces slide upward and downward, respectively, we get the full three inches of elongation. I will put in two over-stitches with the same thread, merely to keep the tendon ends in apposition. Remember, the tendo Achillis spans a greater distance then any other tissue in the body except the trifacial nerve. This stitch merely keeps the ends in apposition. There is no tension on them. The foot now comes up without a particle of trouble, so that we feel perfectly comfortable about this condition.

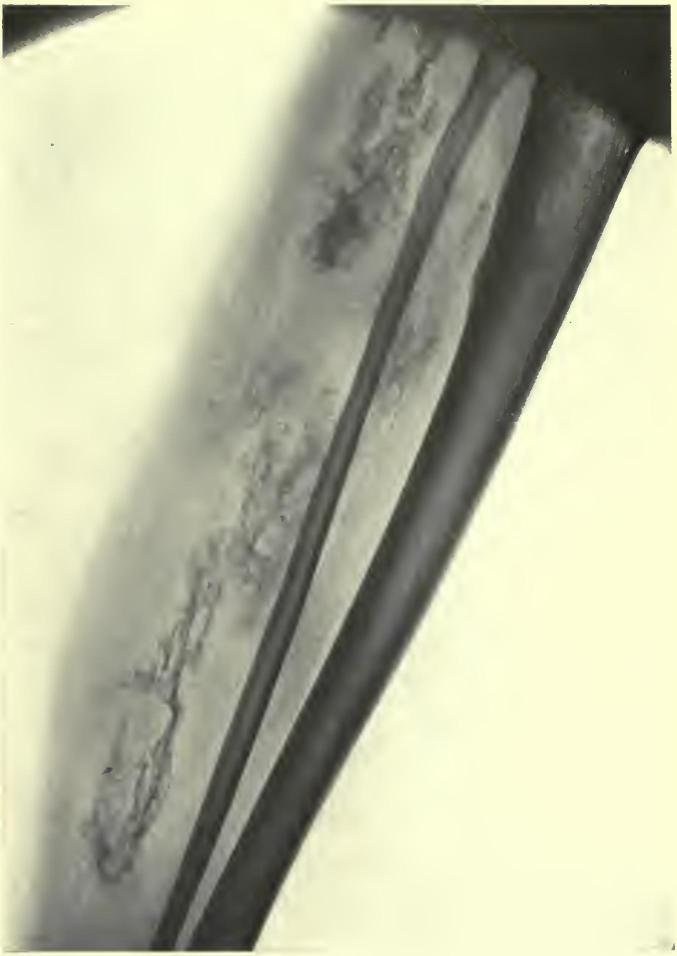

Fig. 103.—Skiagram showing calcareous deposits in veins of leg.

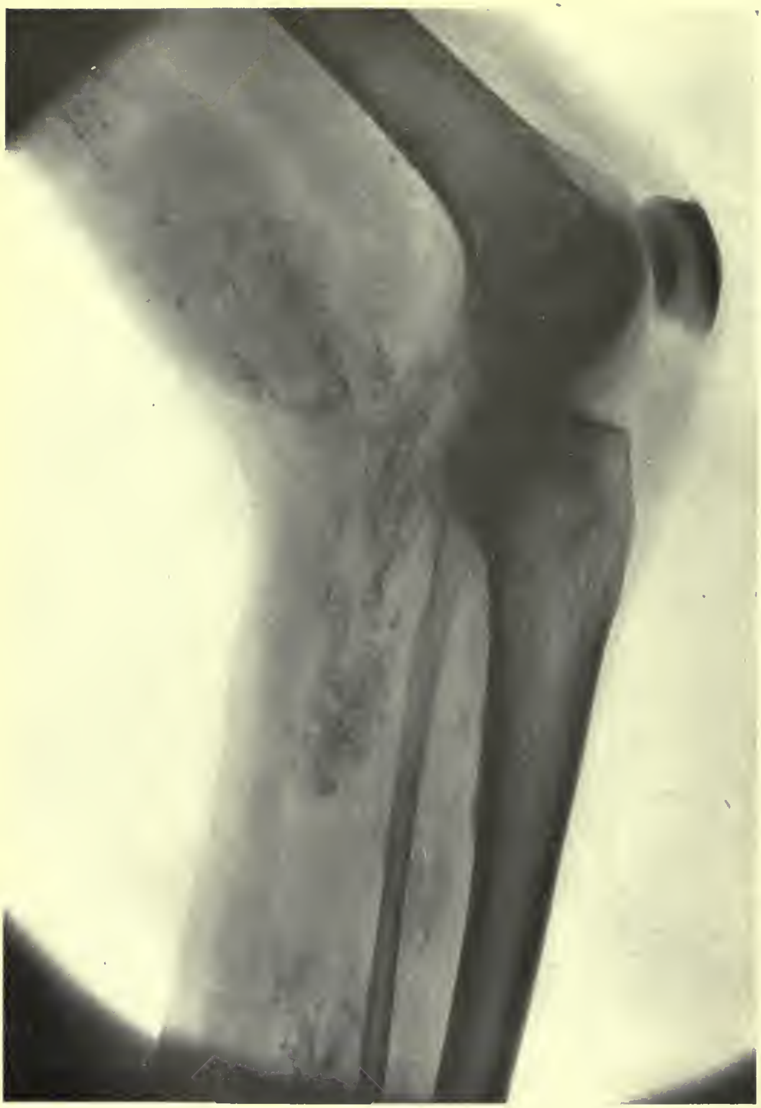

Fig. 104.—Skiagram showing calcareous deposits in veins.

The reason I will not immediately put the foot in a cast in that position is for fear of the posterior tibial artery, which is calcareous, as shown in the skiagram. We may stretch or injure it, but if we will let that stay loose—we will get all the collateral circulation we need to prevent gangrene. Therefore there will be the least likelihood of necrosis of the foot from interference with the circulation in the posterior tibial artery.

In the restoration of these limbs to position they do not come around as readily as this foot did, and the reason that they do not do so is on account of the contraction of the posterior portion of the annular ligament of the ankle-joint. When this is hard to stretch or when it does not tear readily, then I cut clear across it with the tenotome, in the direction of the joint, so that a little pressure fractures it. It is important to do that if you have great pressure, otherwise you are likely to traumatize the ankle-joint in bringing the foot around, but there is no injury done by the scalpel or tenotome except the division of the capsule. You know you are anterior to the posterior tibial artery and you have nothing to fear from that source. The skin wound will be closed with horsehair.

The next thing is to straighten the knee. I let the gastrocnemius give first, because that has to give below and is not going to be cut above. The gastrocnemius can retract to a greater degree than any of the long muscles except the sartorius. When I bend the knee so as to straighten the leg and put tension on the head of the gastrocnemius, it can give from below. That is why I did the tendoplasty on the tendo Achillis first.

Next comes the division of the gracilis, sartorius, semimembranosus, and semitendinosus, the inner hamstring muscles. I am making the longitudinal incision for exposure of the hamstring a little to the outer side, so as not to get it directly into the middle of the popliteal space. Here is one of these angiomatous vessels right in the popliteal space. The tendon is now exposed. This elongation I will make on the same plan as I made the elongation below. Remember that these muscles combine and are attached to the tuberosity of the ischium, so you have to free them as much as you possibly can in order to give the greatest possibility of extending them. I know that this is the tendon by its glistening appearance; in that respect it differs from the nerve, but I will not cut that tendon until I have made the test with the excitor to show that it is not the nerve. I have split the tendon longitudinally, but I have not cut it. Here comes the short muscular tendon where the muscle extends clear down to and forms a cushion on the inner side of the knee, almost to the attachment of the tibia. This muscle must be divided with the tendinous portion that is beneath, from which it derives its name. We must divide that muscle and suture it too. I am making a four-inch elongation. We have the four muscles—the semimembranosus, the semitendinosus, sartorius, and gracilis—coming down here in a bunch. Let us go in and get the nerve, which is still farther back. It shows exactly where you are and you know just what you are doing. While vision is sufficient in this case to guide us right, we cannot overlook the help which the excitor gives us. We will now cut the tendon across above and below at each end of the long incision and suture it in a different manner from what we did the tendo Achillis. The ends will be overlapped just enough to hold them in apposition. That is all. Nature does the rest—fills in the defect and spans the distance, but not so long a distance as it does in the tendo Achillis.

There is the neoplasm or the thrombosed angioma occupying quite a large space. I will extend the leg, but not put on fixation apparatus for fear of compressing the artery. I am going to put on a Buck's extension, which will finally bring the leg into position, but with the least hazard to the patient.

The outer hamstring will be elongated in the same manner. It consists only of the tendon of the biceps femoris.

Just three months ago we escaped having a necrosis of the leg by the closest kind of margin. It was a case of ankylosis of the knee, with the heel almost touching the buttocks. It had been in that condition for thirty years. We elongated the hamstrings and did an arthroplasty on the knee, straightened the leg, but not to complete extension, and put on a cast. The next day the foot was edematous, and the second day it was still more so. The first day it was cyanotic, but the second day it had a little more of a pink color, and the first day we had motion in the muscles of the toes. The third day motion ceased, showing that the axons of the popliteal nerves had been traumatized by the severe extension. About the third day the patient began to improve, and went on to a complete recovery, with only a slight necrosis on the heel. That, I think, was due to pressure from without. Having lost sensation in the leg, she did not know the heel was being pressed on. The leg was saved, however. She has now a straight limb with a movable knee-joint after thirty years of ankylosis. Motion in the knee is very good.

The greatest flexing force of the leg on the thigh is the biceps, and the biceps, you know, is attached to the upper end of the fibula and along its posterior margin. There it stands out, as you see, conspicuously and strong. Remember, the greatest danger in dealing with the biceps lies in interfering with the external popliteal nerve, which comes right along the inner margin of the muscle, turns over the groove on the head of the fibula, and supplies all the extensors of the foot, so that the popliteal nerve has to be guarded carefully in doing this tendoplasty on the tendon of the biceps. If you cut the external popliteal nerve, you paralyze permanently all the extensors of the foot, which the patient needs so much. He needs his extensors more than he does his flexors. There is the angioma. The nerve is right beside it; in fact, the angioma involves the nerve. It is in the nerve-sheath. That is going to come around beautifully with that slight extension. We will bring the leg almost straight, then we will let it relax again so as to protect the circulation for the next two or three days, then we will put on a weight and pulley to pull the leg straight. The same will be done with the foot. It will be kept up only periodically to start with. We put nothing on the foot now but a dressing.

Let the records show that after exposing the tendon of the biceps the external popliteal nerve was exposed and excited with the faradic current, showing it was not injured. The varix was easily demonstrable in the popliteal space—some portions of it hard, some soft and very vascular. This [bending the limb gradually] is one of the nice things which we learned from Dr. Lorenz, doing it slowly and repeatedly and a little farther each time, thus avoiding the danger of creating a fracture and causing considerable trouble.

Visiting Surgeon: Will these deposits in the veins disappear?

Dr. Murphy: The deposits will never disappear. They are typical phleboliths. Phleboliths do not disappear. They are permanent. They are mistaken sometimes for ureteral calculi in making skiagrams of the pelvis.

A dressing will now be applied. The foot is allowed to assume any position. We do not put the slightest pressure anywhere, because we do not know how much these arteries have suffered by that extension.

Note.—Three months after the operation the patient stated in a letter that his leg is straight, that he has full motion, and walks without any support whatever. He does not even limp.

HYPERTROPHY OF THE PROSTATE

HISTORY

Mr. W., aged sixty, admitted to Mercy Hospital April 4, 1912. Came to hospital because of frequent and painful urination and a burning sensation during the act. Family history negative. Contracted lues thirty years ago, followed in four or five weeks by a skin eruption. Hair did not fall out. Had mercurial treatment for one year, and no manifestations of disease since. Had gonorrhea twice, with a urethral stricture following the second attack. Had typhoid at ten, with good recovery. General health during adult life has been good.

Present Trouble.—About twenty years ago, following the repeated use of sounds for the dilatation of a urethral stricture, patient had to get up once or twice at night to urinate. The act was attended by slight burning pain. The pain disappeared in three months, but a slight frequency in urination persisted until about eighteen months ago, when it became worse and patient had to urinate four or five times every night. At first there was no pain during the act, but later, preceding each urination, he had rather severe pain, was slow in starting the stream, and at the end of the act still had a desire to urinate. This condition continued for six months, when retention set in (which was one year ago). A doctor was consulted, and he catheterized the patient. Catheterization was continued twice daily for two weeks, the bladder being irrigated after each evacuation. Following this the patient was able to urinate small quantities at a time, with slight pain, but felt that he never could empty the bladder. Two weeks ago patient again had retention, preceded for ten days by very severe burning pain. For the past two weeks he had to be catheterized twice daily. Has not noticed blood in his urine for the past twenty years. He did, however, see blood after sounds were passed. Patient is extremely nervous. Urine contains some blood and a little albumin.

COMMENTS AND OPERATION

This is an interesting case, and it has peculiarities about it which attract attention. This man has a hard, nodular, woody, and indurated prostate, extending out to the extreme margin of the pelvis. It is a little suspicious of malignancy. It is not very movable around the neck of the bladder. The urine was said to contain some pus and some albumin. He has no evidence of a renal lesion. He is sixty years of age, but does not look over fifty. He is well preserved. He is not the type of man in whom we would look for an enlarged prostate as a senile manifestation, and this is not a senile prostate. He has enlargement of the prostate from an entirely different condition.

If you analyze the lesions of the prostate that bring the patient to you for an operation you have, first, the cases of infection, not because they are more frequent, but because they occur earlier in life. Second, the hyperplasias of the prostate, the senile hypertrophy, as a rule a fibroma, and this condition occasionally undergoes cystic degeneration, causing the formation of small cysts. Third, malignant lesions in the prostate, namely, carcinoma and sarcoma, the former being by far the more common of the two.

Subdividing these further there are, first, the superficial infections of the prostate that occur early in life as the result of a gonorrhea. They occasionally produce an abscess in the prostate, swelling of the perineum, chills, and fever. These patients are intensely sick. Second, there is the tuberculous variety, the disease invading the crypts, slowly infiltrating the gland, and finally appearing in the capsule as a hard mass, with healthy prostatic tissue all around it.

Then there are the general infections of the prostate, not simply an abscess, but a diffuse chronic infection of the gland the result of a chronic Neisserian infection or repeated catheterization. There are diffuse infiltration, diffuse enlargement, caused by an increase in the connective tissue, and hyperplasia of the glandular structures, the latter not being so marked as the former. That extends and usually involves both lobes. In that type of prostate there occur a slow infiltration and fixation of the capsule to the gland tissue, which make it so difficult to enucleate the gland. If you get the case in the early stages of the process, during the infiltration stage of the disease, you can easily enucleate the gland, as you can all freshly infected connective tissue, but when the connective tissue of the capsule has extended down into the gland between the lobules, so that there is formed a dense, cicatricial, and infiltrated mass of tissue, it is very difficult to enucleate the gland. You have to tear it out. It does not come out from the capsule like the ordinary prostate does.

The ordinary hypertrophies of the prostate involving the three lobes are divided, first, into the small fibrocystic prostate; second, the intermediate sized prostate, with irregularities in its lobes, and, third, the very large hyperplastic prostate.

A prostate produces obstruction not in proportion to its size, but in proportion to its interference with the outflow of urine from the bladder by pressure or contraction. A little prostate not larger than two almonds, placed side by side, with an irregular fibromatous mass projecting into the urethra, will often produce more obstruction than a prostate which is much larger. Therefore, when you examine a prostate, the size of the gland is important only if it produces any obstruction to the outflow of urine. There may be a torsion of the urethra from the irregular growth or hyperplasia with contraction of the gland. These are the dangerous ones that are kept under treatment for a long time. There may be a little bit of a lobe between two big ones, but, as I said, the size is not the essential element in all instances.

Interference with the prostate is indicated when it commences to lead to what is known as catheter life. You do not interfere with the prostate solely because it is enlarged, but when the patient reaches the stage when he has to use a catheter constantly to withdraw his urine, we should operate. When a patient has a primary attack, the prostatic obstruction may be caused by a cold.

A metal sound is passed through the urethra into the bladder, to act as a guide during the enucleation of the gland. I will cut through the abdominal wall just above the symphysis, and as soon as I get through the skin and deep fascia, I divide the heads of the recti on both sides, so as to get into the space of Retzius. Then I displace the heads of the recti upward and split the fibers of the aponeurosis in the midline. The peritoneum is next incised, and we see the wall of the bladder. I am pushing the peritoneum well back so as to bring the bladder into the field. Next, we insert into the bladder what we call the anchor stitches, so that the bladder cannot get away from us at any time during the procedure. If one uses traction on the bladder, he does not let the bladder get away so that he has to open up the space of Retzius to locate it again. The bladder will now be opened between these traction sutures, and then we will let the urine flow out.

Passing my finger down into the bladder I can easily palpate the prostate. It is, as I said, hard and infiltrated, mostly on the right side, crowding into the urethra. The slight enlargement we described before operating is there and of the type we pictured. There is practically no enlargement at all on the left side.

Let the record show that the right lobe is enlarged and firm, and that there is a fixation at that position. It is very difficult to remove it. The small gland is much the more difficult to free. The finger only is used to shell out the gland, enucleating one lobe at a time. The sound is an excellent guide in doing this work. There is not much bleeding. Pads wrung out of hot water easily control it. The key to all this work, which was brought out first by Freyer, but was not accentuated by him, has not been fully appreciated by the profession.

Having enucleated the gland, we will put a large-sized drainage-tube into the bladder through the abdominal wound, remove the traction sutures, and stitch the bladder around the tube. A rubber catheter is passed into the bladder through the urethra after the sound has been withdrawn. This catheter is fastened to the prepuce by means of a silkworm-gut suture.

We then close the peritoneal wound in the usual manner, suture the recti muscles back into place with catgut, close the sheath, and then sew up the abdominal wound with deep figure-of-8 silkworm-gut sutures and superficial horsehair sutures. All the structures are restored to their normal position. I put in one stitch to hold the bladder up in place.

To recapitulate: When you have opened the bladder, having first anchored it with the two stitches, the next step is to estimate the condition in the bladder and the size of the prostate. Here it was exactly as we described it before the operation. There was only a small, fig-like central lobe, but large enough to produce obstruction at the trigone. The sound is in the urethra. Passing my finger along the anterior and right margin, I force it into the neck of the bladder or into the neck of the prostate; then I have the anterior urethra at that point, and get in along the anterior margin of the isthmus. After the urethra is torn I swing my finger around under the capsule and on the anterior surface of the urethral portion of the prostate. I then keep swinging to the right, keeping inside the capsule; then back to the midline. I do the same on the left side.

I cover the skin with boric powder instead of bismuth subiodid, because the former is an antiseptic under these circumstances while bismuth is not. The figure-of-8 stitches are tied there over a strip of gauze. Freyer does not leave a drainage-tube in the bladder. We do, and we use the Bremermann constant irrigation with sterile water for about seventy-two hours, depending on the nature of the case. The suprapubic drain will be removed in a week or ten days and the wound allowed to close. If there is any infection present, drainage must be maintained for a longer period of time. The urethral catheter is then also removed. It may be necessary to pass sounds later if there is any evidence of a urethral narrowing or stricture.

As to the indications for operation: The size of the prostate has nothing to do with the indications for operation; the obstruction to the outflow of urine has. The first time he has an obstruction, if it is the result of exposure, the patient is not to be operated on right away. Put him to bed, give him a hypodermic of morphin to quiet him, and the retention will disappear. The moment the man begins to lead a catheter life, no matter how cautious you are as to asepsis, no matter how well the catheter be used, it is only a question of time when the patient will have an infection of his bladder from the use of that catheter. Antiseptic precautions count for nothing. Urethritis, from the use of the catheter, and sepsis of the bladder are bound to occur sooner or later.

In his first article Freyer said he dissected the prostate away from the urethra, and then every practitioner intended to do it himself, and found, as most of us have, that it could not be done. The fact of the matter is that Freyer showed beautiful specimens where he had removed an inch and a half or two and a half inches of the prostatic urethra.

There is no carcinoma in this case. There is merely a hyperplasia of fibrous tissue. It is difficult to get out a hard prostate. Large prostates are taken out by the suprapubic route, by the plan I have followed. We are doing four of these operations by the suprapubic route to one by the perineal. There is less injury done to the urethra by doing the suprapubic operation. If we analyze the cases that are operated by the perineal route and those that are operated by the suprapubic route, we will find that the mortality depends largely on the judgment of the operator more than on anything else. If the surgeon is careful about operating in cases of arteriosclerosis or infection, he is likely to have uniformly good results with either of these operations. If, on the other hand, he operates when there is decomposition of urine, when there is an active infection, the mortality is very likely to be high, no matter what route he selects. The mortality depends on the conditions present with the enlarged prostate more than on the method of its removal.

NEPHROPYELOPLASTY

HISTORY

Miss R., aged thirty-one, stenographer, came to hospital because of recurrent attacks of pain and swelling in the region of the left kidney.

Family history negative. Measles and whooping-cough when a child, with good recoveries. No other illness. Has a slight leukorrhea.

Present Trouble.—Fifteen years ago patient had a dull aching pain in the left side and in the back over the kidney, accompanied by nausea and vomiting. Would not get complete relief from pain on lying down. At that time she never noticed any enlargement during the attack, but there was marked tenderness in left kidney region during the attack. She does not remember any tenderness being present after the attacks at that time. Each attack would last about one hour. No chills; no fever.

Five years after the onset of this trouble she noticed a well-defined enlargement in the left side, just beneath the ninth costal arch, coming on with each attack, and remaining for from two to three weeks after each attack. The patient also noticed an increase in the amount of urine passed immediately after the subsidence of pain. For the past ten years tenderness was very marked during and subsequent to each attack. There is also constantly present a slight tenderness on deep palpation. When questioned regarding tight lacing, the patient states that she did not err. She was unable to wear corsets during each attack, but when effects passed away she is more comfortable when wearing corsets. Attacks are more frequent during last few years and last longer. Patient is now recovering from an attack which lasted two weeks. There is pus in the urine.

COMMENTS AND OPERATION

Dr. Murphy: No casts and no blood in the urine?

The Intern: No, doctor.

Dr. Murphy: There is a very good history. It is practically a classic history of this type of trouble. A typical Dietl's crisis, typical retention of fluid in the pelvis of the kidney, recurring at intervals, first manifested years ago and increasing in frequency and in duration. She had first pain, then nausea and vomiting, not coming on quickly, but after a considerable lapse of time. Then came the manifestation of a tumor in her side. She noticed that tumor enlarging and becoming more sensitive as it enlarged. She has diminished urination during the pain. Finally, as the tumor reaches its climax in size and the pain its climax in severity, and when the reflex symptoms are at their worst, she suddenly has a cessation of the pain, an increase in the quantity of urine, a gradual diminution in sensitiveness over the tumor, a diminution in the size and resistance of the tumor, soreness continuing for a number of days,—eight or ten,—and then a return to the normal condition. She has found that she can lessen the severity of the symptoms by going to bed, lying on her back and elevating her hips, getting her kidney down below the costal arch and getting her ureter straightened out. That is a Dietl's crisis in all its particulars.

As a result of years of distention of the pelvis of the kidney she has developed a chronic pyelitis—she has pus in the urine. She has had no elevation of temperature at any time in these attacks?

The Intern: No, doctor.

Dr. Murphy: This means that she had no virulent infection. What does it all mean? It means a periodic retention of urine in the pelvis of the kidney from any one of the many causes that produce it. The most common cause to produce this condition is an angulation at the attachment of the ureter to the pelvis of the kidney. The next most frequent cause to produce this condition is maldistribution of the arteries: one of the arteries, in place of passing along on the anterior side of the ureter, comes out under the ureter and is attached to the kidney on its posterior surface. The normal anatomic attachment of the large renal veins and renal artery is anterior to the ureter always. As this artery passes around the ureter it compresses and angles the ureter, and as the kidney enlarges the artery is pushed down and becomes tighter and tighter.

Israel, of Berlin, was the first accurately to describe that condition, and his book on the Surgery of the Kidney was the masterpiece of that time. It is based on the clinical study of over 300 cases of renal surgery, and it is the best book that was ever written on renal surgery because it was based on facts, on personal observations made by one man, and I believe that all text-books of the future are going to be written on that basis. That is the proper basis on which to write books, because then you give first-hand information and give it from personal, clean-cut observations at the operating-table and at the bedside.

I cannot feel the kidney now, although I felt it some days ago.

In operations on the left kidney you want all the room you can get. You must remember, when working on the left side, that the diaphragm not infrequently comes an inch or an inch and a half below the costal margin—more frequently below in the male than in the female.

The patient is placed on her right side, with the right leg extended and the left leg flexed on the abdomen. The body is raised at the waist-line by means of sand-bags or a table which may be elevated at an angle in the middle. This brings the kidney well out from under the ribs and throws it up against the body-wall. The incision is extended downward and inward from the outer edge of the erector spinæ muscle at the level of the twelfth rib to the desired length. It must be large enough to give free access to the kidney. The abdominal wall is cut through layer by layer until the perirenal fat presents itself in the wound. This is torn through until the kidney is reached. Remember you are behind the peritoneum.

There is the twelfth intercostal nerve and the ilioinguinal. That you do not wish to cut, nor do you wish to include it in the final closing suture. If you include it in your suture, the patient suffers intensely with pain for several days after the operation until the nerve is destroyed.

Now I commence and look for the kidney. Here it is, below where I went in—and well below. I have planned the incision so as to cause the least possible injury to the muscles. There is a mass that feels hard and firm. I wish to avoid the peritoneum, if possible. That mass should be the kidney. Its capsule resembles the peritoneum very closely. On the left side you are more likely to get into the peritoneal cavity below. The true fatty capsule of the kidney is now visible, and the kidney is shining through. I am well behind it. I must get onto the true capsule of the kidney and then displace the kidney and bring it out. This kidney should be firmly adherent to the peritoneum in front,—very firmly adherent,—because it is so movable and has been sliding around. The kidney is of very great size, with the pelvis enormously dilated. I am well under the upper pole, but I am not under the lower pole. I will just increase the size of my incision downward through the muscle margin so the kidney will vault forward from below. I am trying to get under the lower pole without rupturing the cortical portion. The delivery of this kidney is unusually difficult because of the size of the gland, but with care it is accomplished.

We have here a beautiful pathology. There is a dilatation of the pelvis that has crowded the cortex and caused a dilatation of the cortex. This is a beautiful illustration of the obstruction that occurs from malformation of the ureter the result of pressure by the vessels. The point of obstruction is close to the pelvis. It is a valve-like formation, first described by Dr. Fenger. There are some connective-tissue bands passing along below the ureter. There is no artery here doing the mischief, but the ureter is valved on the side. That is the cause of the trouble.

Will I take out that kidney now? No, there is no occasion for taking out that kidney. It is a good kidney and does lots of kidney work. I will take out all the sac with the valve, and I will reattach the ureter. I will do a pyeloplasty.

You can see now why this patient had a Dietl's crisis, and you can see what the Dietl's crisis did. I mean the condition that produced the Dietl's crisis. First, she had a dilated sac; then, after the sac was as large as it could get, and the collateral supports from the outside made additional pressure on the sac and kept the sac from spreading sideways and downward, it had no place else to go, and it extended up into the pelvis of the kidney.

This large sac has been excised and we can now see how the valve was formed in the ureter, just as Fenger described it. We will now unite the ureter to the stump of the pelvis, but before inserting the last stitch we will pass a ureteral catheter down into the bladder to insure its patency. You will notice that, as I pass the needle through the pelvis wall near the normal renal margin, I get a little bleeding, which we wish to avoid.

Now we commence to do business. I will take this capsule and hold it in this position and show you how I will handle it. Remember I detached that ureter in the fold where I knew the valve was located. Then I cut out the portion of the ureter with the fold. This procedure was described in a paper I read before the Southern Surgical Association. I took out a V-shaped piece of ureter. No other tissue except the peritoneum lives so long and so well as does the ureter.

The next step was to turn out this kidney and take out all the sac clear down to the pelvic margin, until we reached the calices. I amputated that large sac clear down to the calices. Just when you get near the margin of the renal portion of the pelvis you have that venous bleeding and it tells you when you are there—it announces that you are at the limitation of the normal pelvis of the kidney. I closed that sac at the same time that I cut it off until I got down to the place where I reattached the pelvis to the ureter or to the pelvic portion of the ureter. I was very careful not to cut too close to the calices. This is the fourteenth case of this kind in which I have done this operation, and we have 14 patients well.

I made an entropium of the cut edge, just reversing what I do when suturing the peritoneum. If I cut too closely to the lower calyx, I would have a pocket and the operation would be a failure.

Now I let this kidney fall back into its position. We commence to estimate where we are going to attach this ureter. We get this pelvic wall turned out now for the attachment of the ureter. Before I do that I must elongate and enlarge that ureter and make a different opening in it from the one that is in it now. This is the real key to the situation. The ureter must be split where the valve is. I let that portion remain on until I split it. I will sew that ureter as a piece of patchwork on to that pelvis. I am going down five-eighths of an inch below the place where it had valved. I will use a continuous suture of catgut with the knots and the thread outside of the ureter. You can see how that cannot valve or kink again, because the V-shaped patch is put in that ureter below the point where it was formerly obstructed, and it has a flat surface now. The stitches must be put in very carefully, so as to insure perfect approximation of the ureter to the pelvis. The ureteral catheter which we inserted will keep the ureter patent. I pass it out up through the cortex of the kidney and fix it there so it cannot escape. That is the model that maintains the opening in the new ureter until it becomes organically healed or repaired. We will now complete this part of the operation by putting in the posterior line of sutures. The ureter is now securely approximated to the new kidney pelvis.

When we first did this operation—it was an original procedure with us—we had some misgivings; now we have not the slightest misgivings. We know positively that the ureter is going to do its work and that this kidney is going to be saved.

I told you that aside from the peritoneum, this tissue unites more rapidly and securely than any other tissue in the body; that is why we can take such liberties with it. This was a most beautiful illustration of this condition in its superlative degree, with the kidney not materially interfered with, so far as secretion was concerned. There is a great dilatation of the lower pole of the kidney—more than of the upper. There is not so much secreting kidney tissue as normally. I am going to amputate this lower pole, because it has in it very little secreting tissue, and as a large sac it will hazard the final result, as far as this kidney contracting down to its normal size is concerned. I will run a puckering string through and close up this end of the kidney. I am putting the sutures in close to the pelvis, so that any leakage that comes will be siphoned out.

Before putting the kidney back in place we will split its fibrous capsule longitudinally along the convex border, and strip it off down to its attachment at the hilum. Why? To allow the formation of adhesions which will anchor the kidney. Now I will put this kidney back into place. It falls back into position easily. I show you this nerve again—the ilio-inguinal nerve. That must not be included in the suture. First is the catgut closure of the muscularis. We are uniting these muscles accurately because, if we do not, we may have a hernia of the kidney subsequently—a lumbar hernia. I am watching that ilio-inguinal nerve with each and every stitch, so that I will not include it in the suture. Was any effort made to anchor the kidney? No, it will anchor itself. I took off the true capsule, and that will anchor the kidney by the formation of adhesions.

Let the records show that the kidney was greatly distended; that it was a little difficult to luxate it into the incision. When it was luxated, it showed a great dilatation of the pelvis and a dilatation of the calices. The greatest dilatation was at the lower pole—so much so that the lower quarter of the kidney was a sac. The dilated pelvis was first amputated, leaving a selvedge margin to which the ureter could be attached. The ureter was valved at its outlet, and it was this valving that caused the occlusion, and it was this valve-formation that gave the patient repeated Dietl's crises and caused the dilatation of the pelvis. It was this also that caused the dilatation of the lower pole of the kidney, which was exposed most to the pressure on account of the luxation of the kidney downward.

I am lifting up the muscle in the lower loop of the figure-of-8 so as to get it firmly fixed. The skin wound is closed in the usual manner. We attach a large tube to this ureteral catheter, which you remember extends from the bladder upward and out through the kidney substance. The urine secreted by the kidney will flow down the ureter into the bladder, and the catheter will siphon it back again out into a vessel which will be placed at the side of the bed.

We have also placed a rubber drainage-tube into the retrorenal fossa, surrounding it with a strip of nosophen gauze. The sponge count is correct. A very large dressing is applied and kept in place with adhesive.

The drainage-tube will be removed in a week, the ureteral catheter in ten days or two weeks, when there is reason to believe that the ureteral wound has healed. The patient will then be in good condition, so far as the operation is concerned, and can return home as soon as she feels strong enough to do so.

ANKYLOSIS OF THE LEFT ELBOW-JOINT—FRACTURE OF JOINT WITH DEFORMITY

HISTORY

Mr. M., salesman, aged forty-eight. Entered Mercy Hospital February 19, 1912, because of limitation of motion in left elbow- and shoulder-joints. Family history negative. Neisserian infection twenty-three years ago, with no apparent ill results. Was a very heavy drinker eleven years ago, and took treatment for this habit. Has used no alcoholics for the past nine years.

Present Trouble.—November 21, 1911, he was in a railway coach which rolled over an embankment. He was thrown about the interior of the coach with great force, and though he did not entirely lose consciousness, he was badly stunned. He was able to walk out of the car immediately, but found that his left arm hung limp and felt numb. He was taken to a hospital, where it was discovered that his left humerus was fractured at its upper portion, but not compounded. The bony structures of the left elbow were fractured and compounded, the olecranon being driven out through the skin.

The wound was cleansed with tincture of iodin. He states that it never discharged pus. A cast was applied, which he wore for twenty-five days. When it was removed, the shoulder-joint was nearly immovable. Motion at elbow was limited. The forearm could not be flexed or extended in an arc of more than four inches. Forearm was held almost at an acute angle. The movements of hand and wrist were not affected in any way, save for some limitation of pronation and supination. Muscles of arm and forearm are flabby and atrophied.

Examination of heart and lungs revealed nothing pathologic. Urine, normal; temperature, normal.

COMMENTS AND OPERATION

This patient had an impacted fracture of the upper end of the humerus, which has already fully repaired. That the doctor who took care of the case in the beginning did splendid work is evident from the fact that although the patient had a compound comminuted fracture of the elbow-joint,—wide open,—the doctor kept his hands and instruments out of the wound and the result is this primary union. That is something attained worth while under adverse circumstances. He has saved this man from much trouble which he would otherwise have had. At that time the arm was swollen, was severely injured, and the doctor was unable to outline the bone, and even if he had been able to outline it, I do not know that he could have done very much. The fact was that when the patient struck the point of the elbow, the olecranon was driven through the articular surface of the humerus, carrying with it the internal condyle, and dislocating it upward and inward; the outer condyle of the humerus was driven down to or through the skin. The process of repair went on, and there is now a fibrous union of the internal condyle, with the articular surface of the ulna. The external condyle is protruding away below the level of the posterior surface of the humerus. The bones of the forearm were pushed upward, forcing the external condyle almost, if not quite, through the skin. We have here a splendid result, so far as asepsis is concerned.

He also luxated the head of the humerus. There is an impacted fracture of the neck of the humerus. He has bony union, however, and fairly good position, so that it should not be disturbed. The tip of the acromion and the tip of the coronoid process bear a proper relation to each other, and he has there an articular surface without ankylosis.

It is impossible for us to elongate the internal condyle again or to bring it down to the level of the external condyle. What we must do, therefore, is to shorten the external condyle and thus bring it up to the level of the internal condyle, and make it of such a shape that the head of the radius can slide around the external condyle, preventing the head of the radius from becoming

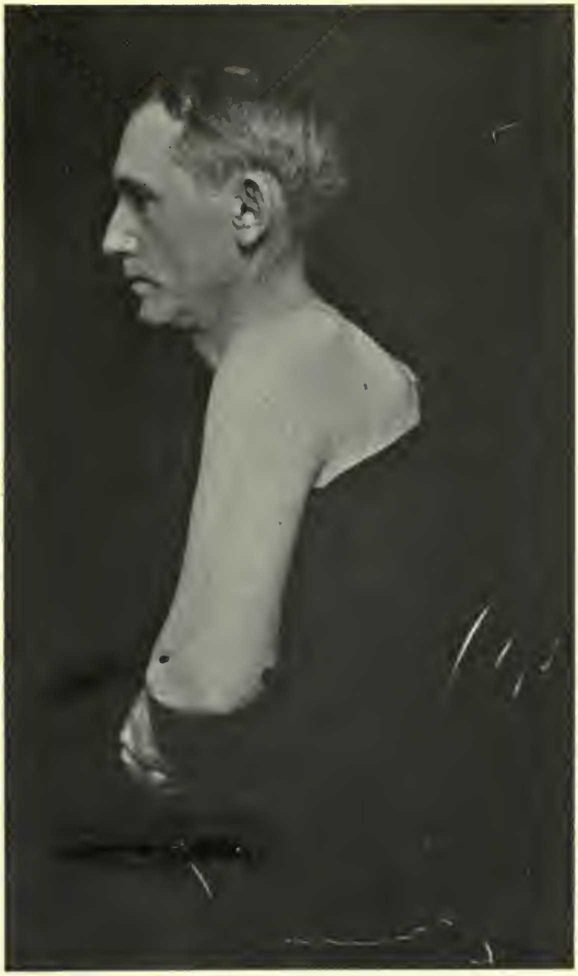

Fig. 105.—Photograph showing deformity of elbow.

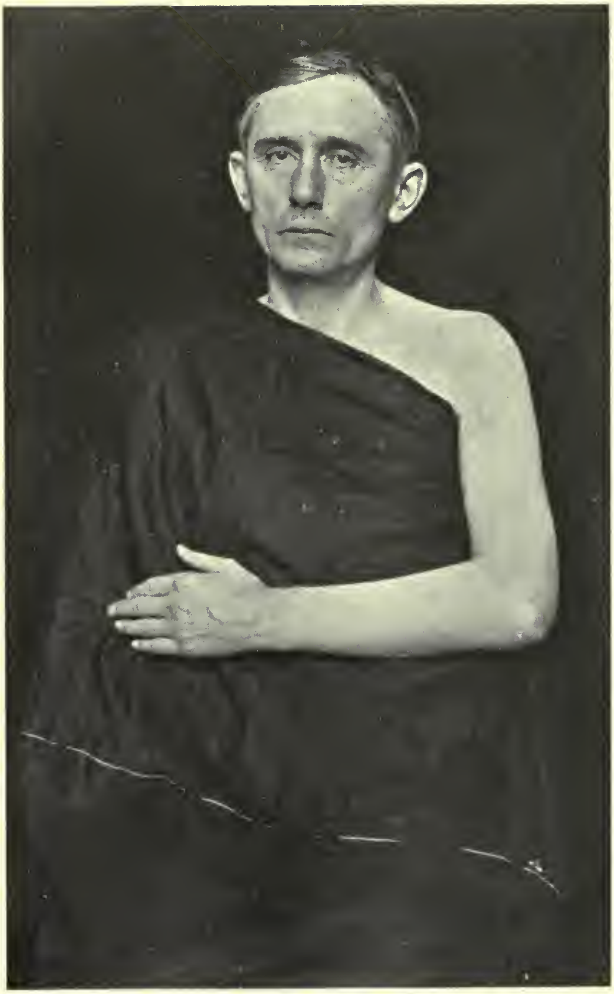

Fig. 106.—Photograph showing fixation of elbow.

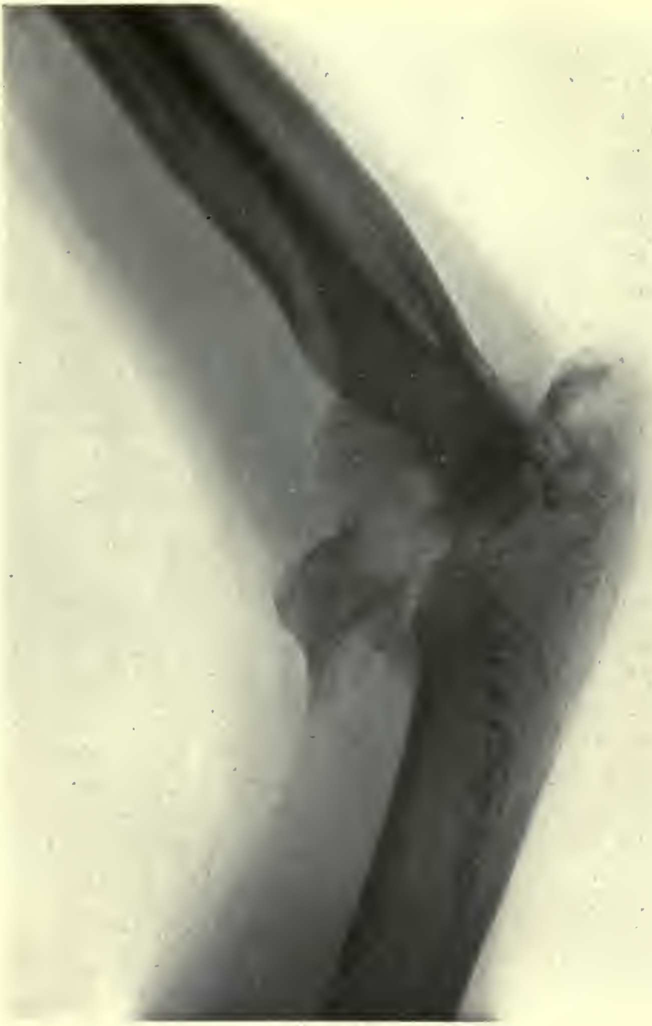

Fig. 107.—Skiagram showing fracture. Marked displacement of fragments.

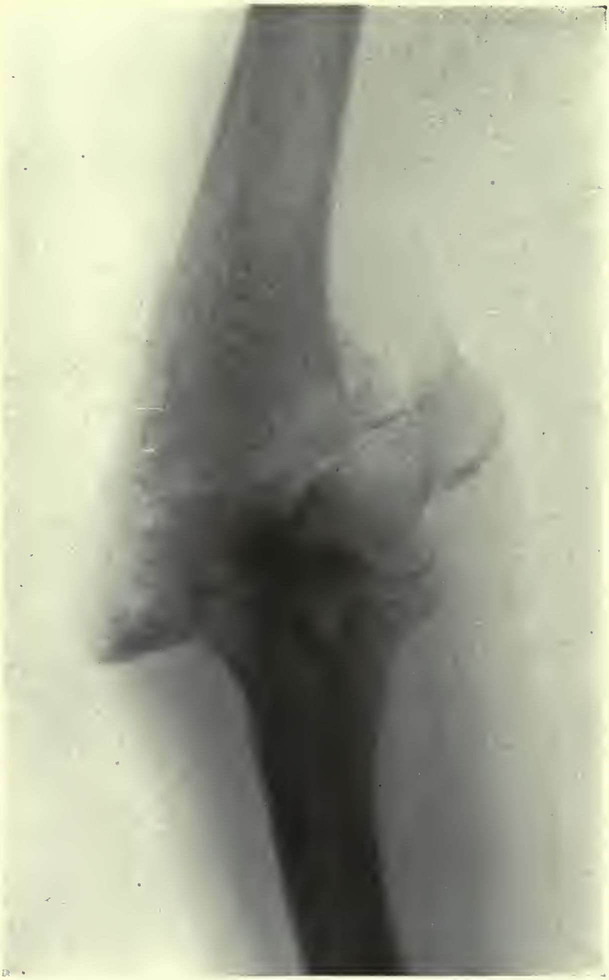

Fig. 108.—Skiagram showing fracture of elbow-joint through the condyles.

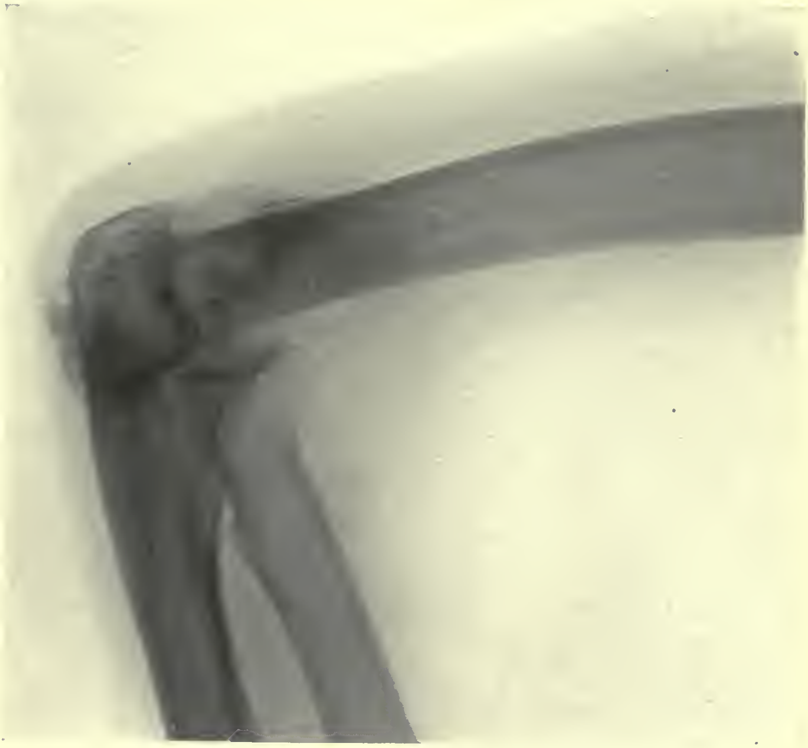

Fig. 109.—Skiagram showing deformity.

agglutinated to the freshened articular surface, which we must make on the anterior surface of the humerus by interposing a fascia and fat flap. In addition to that, if, when we flex the arm, the coronoid process impinges against the anterior surface of the humerus, we must correct that. If the coronoid fossa is gone below, we will either have to take off the tip of the coronoid process so as to get it to come around farther, or make a cavity in the lower end of the shaft of the humerus to permit the coronoid process to go in and not interfere with motion. We will do whatever is indicated when we get in there.

This is a complicated condition. He has been saved from an infection which would have obliterated the synovial surface in the joint and resulted in an ankylosis. Therefore we do not have to do a typical arthroplasty, only a partial one, covering the lower end of the external condyle only, in place of covering the lower end of both condyles of the humerus. The line of action is different from that which we have followed in some of the other cases we have had, because of the ankylosis we encountered there. It is usually an ankylosis the result of infection and inflammation, and not an ankylosis that is due to trauma, as in this case.

It is difficult to interpret the skiagram in this case. The anatomic surface of the articular end of the ulna has not been disturbed. That is the first thing. The internal condyle is broken off from the shaft of the humerus and displaced inward and upward. There is limitation of motion, but not a bony ankylosis. The ulnar nerve is not injured, but it is displaced by a splinter of bone which lies between it and the fractured internal condyle, showing clearly that the relation is not maintained. We must do something to permit this arm to come down. The principal reason the arm does not come down now is because there is an impingement against this external condyle, so that we shall have to take off a portion of it. That is proposition number one. Whether we shall have to produce a new cavity for the tip of the olecranon, or take off a portion of the tip of the olecranon, we will have to settle later

I am going to make my incision on the back of the arm to the inner side of the olecranon, so as to get sufficient tissue for the interposing flap. When we rotate the radius, we find that it is situated away above, instead of almost on a level with, the condyle. The incision is about five inches long. It extends through the skin and fascia, and then the field of operation is exposed. The musculospiral nerve is in view, but we do not expect to get near it. We will, however, be obliged to stretch both the ulnar and the median nerves when we put this arm through the full latitude of motion. We have had three cases of paralysis following this stretching, none of them permanent, however. The paralysis disappeared in all of them after a time.

We will now expose the external condyle of the humerus by dissecting outward, undermining and retracting the skin-flap so as to get a good view and provide easy access. There is the articular surface for the radius on the humerus. The head of the radius is not short at all. It has gone backward. It is fractured. Next, we will dissect inward so as to expose the internal condyle and get our landmarks. I will take off a little of this external condyle, which impinges against the head of the radius, and straighten the joint line. That will make practically a new external condyle and a new articulating surface for the head of the radius—and it is here that I shall interpose a fat and fascia flap to prevent the occurrence of an ankylosis. I provided originally for a sufficiently large flap, which I will take from the outer layer of the capsule and the fatty layer of the deep fascia. I have made it of full size, so that I can insert it over the articular surface, so that there will be no tendency to bony union. These two condyles—the external and the internal—should be on the same level with each other, and the anatomic conformation of the articular surface must be preserved. Of course, the internal condyle will not be disturbed in any way. Therefore, we are really shortening the arm, and while he will not have a good-looking elbow, he will have a good joint, with full range of motion.

The next step will be to cover all that raw bony surface, so that none of it will become adherent to the radius or fixed in any way. I am going now to transpose the fascia so as not to interfere with the contractile power of his triceps. I will cover these

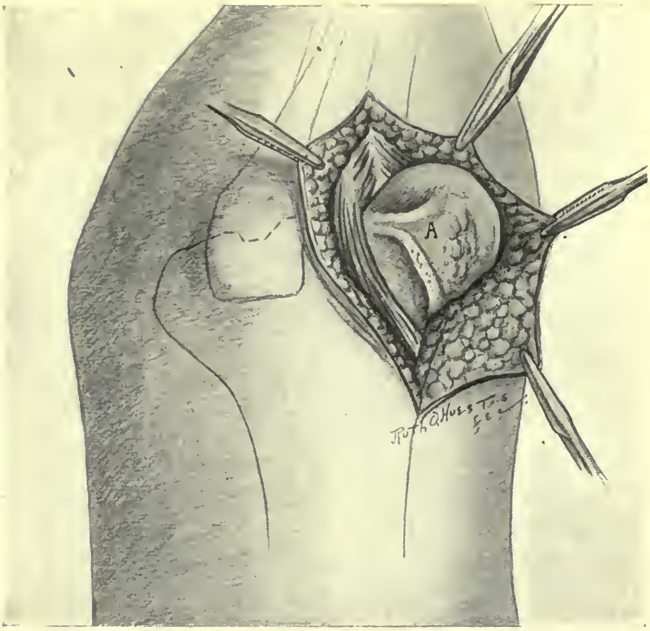

Fig. 110.—Hypertrophied external condyle of humerus (A) exposed.

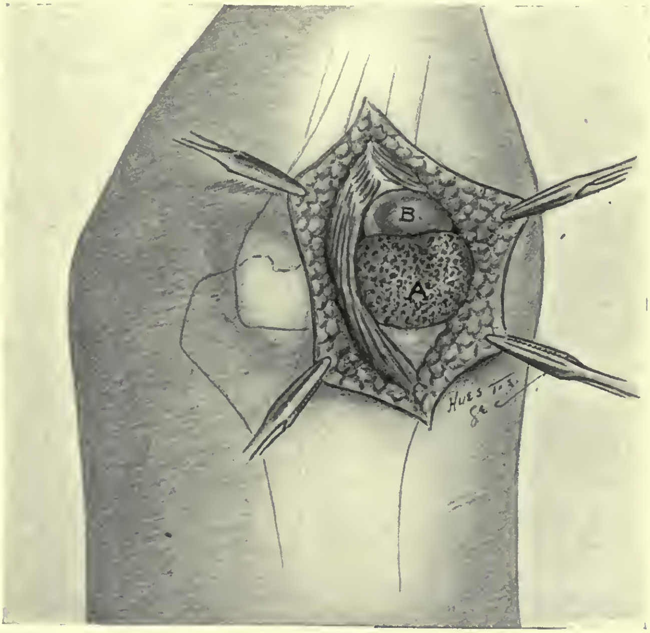

Fig. 111.—A, Redundant condyle chiseled off; B, head of radius.

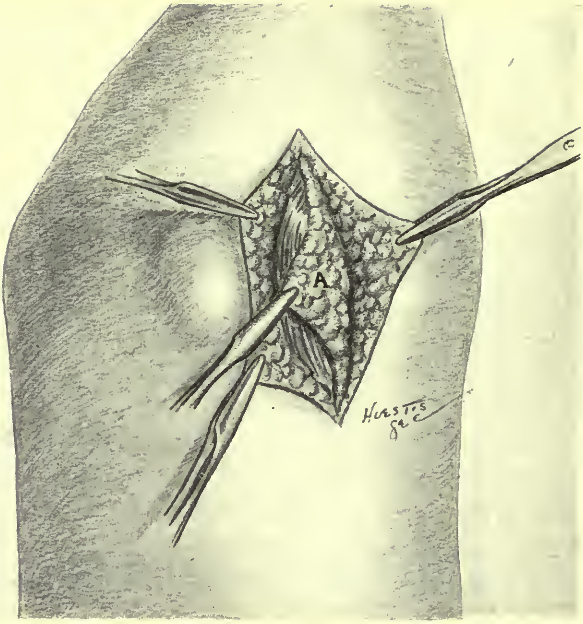

Fig. 112.—Flap of fascia and fat (A) drawn over condyle.

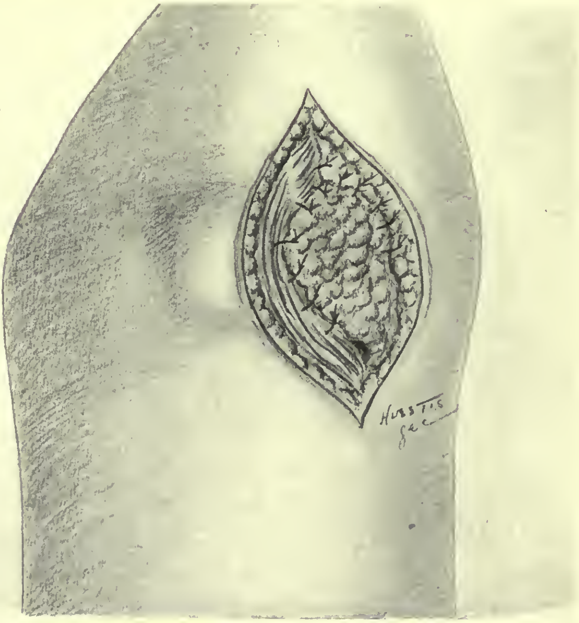

Fig. 113.—Fascial flap sutured in place.

exposed surfaces in such a way as to bury the head of the radius. We do not have to take off the tip of the coronoid process nor the tip of the olecranon. They both came out in good position, without the hazard of impinging, after our remodeling work on the humerus, so that we are safe in letting both remain as they are.

In order to get full extension following such a trauma as occurred in this case one must either shorten the upper end of the radius or remove the obstruction that is produced by the condyle. When the head of the radius has been luxated and has become locked, one of the classic procedures has been to remove the head of the radius, shortening the bone down to the neck. That reduces the strength of the arm very materially, although technically it is not a difficult operation to perform. A procedure that does less injury is reduction of the external condyle. The anterior half of the external condyle can readily be removed and not interfere with the function of the joint in the least, or reduce the strength of the forearm. That is done in exactly the same way as we did here, except that only the anterior half is removed, so as to permit the head of the radius to slide along the articular surface of the external condyle, and in that way getting a full extension. You can see that we have here, without forced extension, a full and free range of motion. The flap is sutured in place with phosphor-bronze wire, such as we always use for this work. There must not be any pressure on the flap or it will die, and the object for which it is put here—to prevent ankylosis—will be defeated. The skin incision will be closed with horsehair and dusted with bismuth subiodid powder. There is no danger here of paralysis of the nerves, and there is no reason why we should not obtain a good final result in this case. The cases in which we have had paralysis, as I have mentioned before today, were annoying in the length of time it took to get restoration of function—sometimes many months. See the ease with which the arm comes down and goes up! There is full motion without any effort. It will be dressed in the flexed position and placed in a plaster-of-Paris cast. This cast is made in the usual manner, being split after the plaster has set, so that it will not constrict the arm when the tissues swell. The internal condyle was not disturbed at all. Elongation of the arm by bringing the internal condyle down on a level with the external condyle was an impossibility at this time.

We use antiseptic gauze dressings in these cases. The first dressing consists of 5 per cent. phenol gauze. The antiseptic dressing is put on for the protection of the secretion that escapes from most of the bone operation wounds. Unless you protect these wound secretions which go into the dressing, the air comes in above and below where the dressing is soaked and decomposition occurs. It is a saprophytic decomposition. This saprophytic decomposition sets up a pyocyaneus infection or a saprophytic infection, and that may extend along that wide zone that has bloody secretion in it and cause infection of the wound. We resort to the antiseptic dressing for the protection of the secretion that escapes between the stitches. You see the reason for using carbolized gauze: it prevents the secretion from acting as a culture-medium.

If that flap should necrotize, we will do a plastic operation, but we are not in the least afraid of that. We have had only one case of bony fixation following operation, and that was of the elbow. It was a case of bony ankylosis. The usual arthroplasty was done and successfully. However, the patient, a very neurotic female, would not permit of any manipulation of her arm afterward. The result was that the elbow again became ankylosed, but not because the operation was a failure.

In this case the cast will be removed in ten days or two weeks, the stitches taken out if the wound has healed, and passive motion begun. After each manipulation the cast will be replaced for a while until it can safely be left off, after four or five weeks. The patient is encouraged to secure elbow motion, so that he will have a useful elbow. [When seen last, three months after operation, patient had a very good range of motion. He could flex the arm to better than a right angle, and extension was almost complete.]

TUMOR OF THE ABDOMEN—RETROPERITONEAL SARCOMA

HISTORY

This patient is a physician, fifty-three years of age. He entered Mercy Hospital March 11, 1912, because of severe pain in the lower lumbar region, a high fever, and great prostration. There has been no malignancy, syphilis, or tuberculosis among his relatives. His health has been excellent up to the onset of the present trouble. At the age of nineteen he had scarlet fever, followed by albuminuria for eight weeks. He was attended by Dr. Osler, who assured him that his recovery was perfect. At the age of twenty-three he had typhoid fever severely. Eighteen years ago he suffered much from very severe headaches and nausea. He, a physician, states that this was migraine, and that his mother had similar attacks. He denies syphilis.

In 1894, when thirty-five years of age, he began to have repeated attacks of intense colicky pains in the region of the gall-bladder. Vomiting always followed rapidly, and the next day a deep jaundice would appear, lasting for from three to five days. The pain would come on very violently and very suddenly. He never had chills or fever. For thirteen years he had these attacks and became a victim of the morphin habit, often taking as high as 12 grains daily. He dreaded operative treatment, always delaying it hoping that each attack would be his last.

April 9, 1910, he was operated on by Dr. Murphy. The findings were as follows: Gall-bladder, duodenum, stomach, omentum, and hepatic flexure of colon all were adherent. No pancreatic abscess. There was one large stone in the cystic duct, and the common duct was full of stones which could be moved back and forth into the cystic duct. Stones were removed from the ducts, each of which was the size of one's finger. A T-shaped tube was inserted, passing into both ducts.

No evidence of acute sepsis was present. Three rubber drains were used in gall-bladder, common duct, and peritoneal cavity respectively.

After the operation he recovered without incident and left the hospital May 15, 1910. For one year he enjoyed perfect health, gaining 70 pounds in weight and ceasing the use of morphin.

In March, 1911, a hemorrhage occurred into the retina of his left eye. A leading physician of St. Paul, Minn., diagnosed kidney disease, having found a blood-pressure of 260, but nothing abnormal in the urine. He left Minnesota, going to the milder climate of Florida within two months following his retinal hemorrhage. He adopted a non-nitrogenous diet, and for ten months felt very well, though the blood-pressure remained between 180 and 200. In the latter part of January, 1912, he became suddenly very ill, with a high fever, delirium, and a severe diarrhea. The fever often rose to 104° or 105° F. The urine never contained blood-casts nor was it increased in total amount. The stools were persistently diarrheic, but never were copious, offensive, or pale, and never contained undigested meat-fibers, fat, or visible blood. At first a diagnosis of dengue was made by able men. The prostration was great from the first. He had no pain until two weeks ago. He states that at this time an intense boring pain appeared suddenly in the back, just above the posterior iliac spines. It seemed to be almost in the middle. It was entirely unlike any pain he had had associated with his gall-bladder attacks, and more severe than any pain he had known. It has persisted with remissions up to the present time. He has used morphin freely since its appearance. He has not been jaundiced, nor has he had emesis during this sickness. He has lost fully 50 pounds in weight during the last six weeks. On palpation of the abdomen a mass could be felt in the position of the tail of the pancreas. It pulsated, but this was not of the expansile type. Temperature on entering the hospital was 101° F. On the morning of operation, 99° F.

Laboratory Findings, March 12, 1912.—The urine contained a moderate amount of albumin by ferrocyanid test only. It contained no sugar. One granular cast was found. Six red blood-cells were found in two slides of the centrifugalized urine. Only an occasional pus-cell was present. Chemical tests for blood in stools failed. The Widal test was negative. Blood count: Leukocytes, 10,600; differential count, polymorphonuclears, 74 per cent.; small mononuclears, 23 per cent.; large mononuclears, 2 per cent.; no eosinophiles were found in a count of 200 cells. Erythrocytes, 4,116,000; hemoglobin, 70 per cent. Blood-pressure, 184 mm. of mercury.

COMMENTS AND OPERATION

As you see, there is a very peculiar history. He went along all right after the operation for gall-stones, when he saw a doctor who made some kind of an unfavorable prognosis because of pain in his abdomen. I do not know what it was. Immediately after he had this unfavorable prognosis made he went back to the morphin habit, which he had contracted previously, taking hypodermic injections, and he took considerable quantities of the drug. Then he lived in the South, a different type of life, doing nothing, resting—when suddenly he had an elevation of temperature. How much pain he has I do not know. He cries out all the time, but whether this is morphin craving or whether it is real pain it is impossible to say. We did not, however, cut down his morphin enough for him to have pain.

This mass to the left of the midline is extremely sensitive. It does not appear to have expansile pulsation. His urine findings are not in favor of an infection in his kidney which might cause this condition. This mass is thought to be his left kidney displaced downward, which puts it in the position of the tail of the pancreas. The patient up to the onset of his illness was a very hard worker and had an influential position in his community up to the time of contracting this drug habit. He did a thing that a doctor should never do—take his first hypodermic of morphin if he has a pain, especially if he has a chronic trouble. Often doctors give hypodermic injections to women during menstruation in fact, doctors give them so often that the thought makes me creep. The frequency with which anodynes and stimulants are given to relieve pain during menstruation is appalling, and the number of victims it leaves is enormous. A man should be held responsible in some way, it seems to me, for starting patients on this downward course; to many it is worse than death in the end, and I feel we cannot be too emphatic in condemning the practice of doctors taking hypodermic injections of morphin themselves or giving them to their patients for chronic troubles.

We will open the abdomen in the midline in the usual manner, making the incision extend between the xiphoid cartilage and the umbilicus. There is the umbilicus; when this is lifted there is the mass. It is hard and pulsating. There is nothing to indicate that it is an aneurysm of the renal artery. Why did he have this high temperature? He had a diarrhea, and *following* the diarrhea he had his pain—not so much during the diarrhea. I am going in behind that mass. It is movable and resting on the vessels. It pulsates, but there is no bruit palpable. I want to examine this condition above to see if his temperature elevation has any definite relation to his old condition. There is no evidence of tuberculosis in his family. The mass is not the kidney. It is in the region of the tail of the pancreas. It is behind the mesentery, and low down. It is not in the pancreas. I am displacing the small intestine so as to get a better exposure of this mass. There it is. It is a sarcoma—and of the pulsating variety. There is the mesentery of the intestine on the side. To the right is the abdominal aorta.

Visiting Surgeon: What is that mass on the other side, doctor?

Dr. Murphy: There is no mass on that side. That is higher up, and did not originate in the pancreas: it originated behind the peritoneum—retroperitoneal, because it is on the under side of the great mesentery, and it has elevated the posterior peritoneum on the under surface of the intestine and the latter is adherent to it. The abdominal aorta is to the right of the neoplasm, and the neoplasm pulsates like an aneurysm. It is not expansile. There may be elevation of temperature with sarcoma without infection, even a high temperature. I called attention to that many years ago.

The first case of this kind I saw was in Dr. Fenger's service during my internship in the Cook County Hospital. The patient had a trauma of the knee. He worked that day and the next day, and on the third day he had a high temperature. When he came to the hospital that night he had a temperature of 104.5° F. It remained as high as that for many days. It looked to us as though he had an osteomyelitic infection in the bone near the knee. Dr. Fenger was not satisfied about it. In a few days the mass began to pulsate; then a diagnosis was made by Dr. Fenger of an acute aneurysmal type of sarcoma. In a few days more a distinct bruit could be heard over the internal condyle of the femur, and in a few days more his leg had to be amputated at the hip. There was a distinct aneurysmal formation.

This case was the first to call my attention to the high temperature which may be associated with acute sarcoma. Our patient here began in this way. His first symptom was the high temperature, no particular pain; then pain in his side manifested itself and it became rapidly worse.

VISITING SURGEON: How would you explain his retinal hemorrhage and high blood-pressure, doctor?

DR. MURPHY: I presume it was the usual high blood-pressure accompanying a chronic infection. For years he had an infection in his gall-bladder. Most high blood-pressures are sequences of some previous infectious condition, usually of a chronic type. His gall-bladder was infected for years before he was operated on the first time. His morphin habit interfered with his assimilation, and the retinal hemorrhage is believed to be due to that. It is not at all settled as to why people get high blood-pressure and get it early, any more than it is at all settled why some races are more prone to arteriosclerosis than others. The Hebrew race is predisposed to arteriosclerosis, and it occurs very early in life. I have seen it as early as thirty-two. This is not at all the condition with the Germans—not at all the condition with the Americans, though they get it much earlier than the Germans do. The claudication manifestations, for instance, that come with the ischemias of the leg, and especially flat-foot, are practically a Semitic disease. We see it very frequently in these people.

I remember one man who had an arteriosclerosis involving his right lower extremity first, and then the left. Finally double amputation had to be done. I made one of them, and the other was made by another surgeon. He also was treated for four years for left renal colic by very able men. He had a recurrence of the renal colic, and then I saw him in consultation with Dr. Sippy. Dr. Sippy asked me to operate on him for an obstruction in his left ureter, and I concurred in the diagnosis. He had passed no urine for six days preceding the operation. He had great pain in his left side, colicky in character, resembling renal colic pains, such as he had had many times before. We looked him over and we concluded that he had a renal calculus with sympathetic anuria in the right side, as described by Dr. Bevan. I cut down on his left kidney and exposed it; lifted it out and peeled off its capsule. It did not bleed. I cut into it and it did not bleed. It was necrotic and dead. He had an arteriosclerosis of his renal artery, with spasmodic closure which gave him the symptom of renal colic, and then his kidney, for want of nutrition, became gangrenous. He died nine days from the onset of his disease from uremia. Autopsy showed that he had a complete occlusion of both renal arteries, the result of an arteriosclerosis with secondary thrombosis. The occlusion was produced gradually, caused by an endarteritis obliterans, which is the type of disease these people have. It was the cause of his death.

I believe the flat-foot which these people have frequently is the result of an ischemia—probably in a large percentage of the cases. You will find that many of these people suffer from flat-foot, claudication, and inability to walk any distance without pain.

This man, instead of having the gastric crises that are often a manifestation of arteriosclerosis, had a renal crisis which comes with endarteritis obliterans.

It is very interesting to follow that class of cases. Here is a comparatively young man, fifty-three years of age, with an arteriosclerosis, although this condition is not uncommon in the young. I remember the case of a prominent merchant of this city which was another one of that type—apparently perfectly well, always attending to business—a high-tension man. He had to be operated on for acute appendicitis. He stood the operation without a particle of trouble. In three days he was on a back-rest. He said to the nurse, "Will you kindly move those flowers from that position to that side. I think they will look more beautiful. Madam will be here in a few moments." The nurse lifted the flowers, took them from one window to another, a distance of not more than five feet, and when she turned around he was dead. Autopsy showed an endarteritis obliterans involving his coronary artery. He had a myocarditis of ancient origin and white streaks all through his myocardium.

Another man about fifty-one years old, head of the greatest bond house in the West,—a high-tension man,—had endarteritis obliterans or arteriosclerosis of his coronary artery. He had repeated short attacks of angina, always occurring just at bedtime. In some of these attacks the pain became more severe. In one of these he collapsed, became cyanotic, and died some fifty hours afterward. I examined his heart in this period and the expression which I used at that time was that the heart seemed to be in China, it was so very weak. During the spasm he had a thrombosis of his left coronary artery which caused his death. That accounted for the feebleness of the heart-beat during the time intervening between the onset of the thrombosis and his death. The only lesion was in the coronary artery—just one single artery. He had exactly the same manifestations that all these cases get. For six months he had pain at night before going to bed, and always at the same hour—so characteristic of these lesions; always pain in his chest and difficulty in breathing. He came in from the theater one evening, and while taking off his clothes he had a sudden severe cramp and fell to the floor. Usually the patients die instantaneously.

We shall not attempt to remove the tumor in this case, because of the absolute futility of such a procedure. The patient's life would not be prolonged. The peritoneum will be closed with a continuous catgut stitch, everting the cut edges so that the inner smooth surface will be in contact. Figure-of-8 catgut sutures approximate the deep and superficial fascia, and horsehair closes the skin wound. No drain. Bismuth subiodid powder and a collodion dressing protect and seal the wound. The stitches will be removed about the tenth or twelfth day. [About eight or ten days after the operation this man complained of a sudden severe pain in his abdomen. He died within three minutes—before any one could get to him. I believe there was a rupture of the sarcoma. An autopsy was not permitted. —J. B. M.]

CONCUSSION OF THE SPINE WITH IMPACTED FRACTURE OF THE VERTEBRÆ

(Case Shown March 14, 1912.)

On January 17th 200 pounds of meat fell on this man's shoulders while he was in the stooping position. The shock rendered him unconscious for half an hour. When he regained consciousness he found that he could not walk, but he says that when he touched his legs he could feel them. That means that sensation was not paralyzed. He had paralysis of motion for thirty-six hours, and then he began to move his legs, and gradually has moved them more and more since. This is a beautiful case of concussion of the spine with impacted fracture of the twelfth dorsal and the first and second lumbar vertebræ, causing this apparent kyphosis, and resulting in concussion of the cord. Now, because he has only paralysis of motion and not a paralysis of sensation, we know at once that his legs will regain their motive power. The cord has suffered only a contusion. The trouble is in the zone of the conus, and not in the true cord. The axons are covered with neurilemma outside of the cord, and are fully capable of regeneration after contusion, concussion, or division if they are contacted, while if the lesion involves that part of the cord where the axons have no neurilemma, then there is no capability of restoration. Once the true cord is cut, it never regenerates, no matter how accurately it is approximated. This man is having a softening or osteitis traumatica of his vertebræ, and unless he is given mechanical support to his back he is going to bend over forward. While the process of repair is going on, until there is complete ossification of the bone, he should have a mechanical support to hold him straight, and that mechanical support should be a plaster cast.

This man did not get a metastatic infection with tuberculosis with his trauma, as he would have if he were a child. A child that receives that kind of an injury always has tuberculous osteitis; children not infrequently get tuberculous osteitis resulting from trauma, which begins in the anterior or posterior portion of the body of the vertebræ, depending on the position in which they are struck. This man was struck on his shoulders; the anterior parts of the bodies of his vertebræ were injured. If he had fallen on his face and had been doubled backward, he would have a lordosis. I remember a patient who came to me for diagnosis. While walking one night in a stable he caught his foot in one of the lines across the pathway. He was thrown forward and struck on his hands. He got up and did his work. He got a pain in his back, but paid no attention to it. In five or six weeks it began to get worse. Liniment was put on for what was called "lumbago." He went on and was treated and treated, and finally, after a year and a half, he came here. He had a typical lordosis. An abscess developed on the side of his spine, deep in. We aspirated and gave him tuberculin. He went through the regular treatment for tuberculosis of the spine; his case was a falling forward and doubling of his spine backward. The injury in this case was posterior.

This man will have to wear a plaster-of-Paris cast for a long time. If it is taken off early, his condition will get worse. His paralysis was mild. Occasionally you have another type develop in this same relation—where the individual strikes on the buttocks, side, or on the shoulders. The effect is the same. You cannot have an immediate severe trauma of the spine without paralysis, and paralysis can commence to develop in ten days or two weeks, or three or four weeks—that is, complete paralysis.

I recall a case I saw three weeks after the injury. In five weeks he had complete paraplegia—bowels, rectum, and everything. We made a favorable prognosis, and in fourteen months he was walking; in two years he was back at his work and has been there ever since. That occurred in 1886. It was one of my first cases of the kind and called my attention to the significance of that type of injury.

TRAUMATIC EPILEPSY; DECOMPRESSION

HISTORY

This patient is a coachman, forty-six years old. He entered Mercy Hospital February 29, 1912, because of partial paralysis of right side of body, speech difficulty, and periodic attacks of unconsciousness, resulting from a blow on the left side of the head. He denies the existence of syphilis, alcoholism, epilepsy, or other nervous diseases among his relatives. He denies all venereal diseases in self. His past life has been normal in all respects and free from disease or operations.

Present Trouble.—In September, 1910, the patient was struck on the left side of the head, four inches above the ear, by a five-pound hammer. The blow was glancing, and he was not rendered unconscious. He was able to walk away from the scene of injury. About one hour afterward he found that he could not move the muscles of the right side of the body as usual. He thinks the power of movement in right arm was entirely gone, and that he could move the right leg but little. The doctor told him that the skull was fractured and scalp wounded slightly. Four hours after the injury he was operated on, but does not know what was done save that the skull was opened. Five weeks later he could move the hand a little and movement in face was much improved. All movement became better very slowly, and he could do his work and walk about, though his leg dragged somewhat and he could use his arm to only a limited extent. He could bring it up to a level with his shoulder. The leg seemed less affected, according to his statement. Following the injury his speech entirely disappeared, but after the operation he could talk a little, but with great slowness, especially at first.

November 15, 1911, he suddenly lost consciousness and thinks that others said he was asleep, and could not be wakened for fully half an hour. Afterward he felt dizzy and had a queer feeling in his head for twenty-four hours. He knows nothing at all in regard to convulsions or muscular spasms at this or at any other time.

February 26, 1912, he knows that he again "went to sleep" and could not be awakened. He remembers very little about it except that he had a queer, dizzy feeling in the head. He never vomited or had severe headache at this or at any other time. Temperature is normal; urine, negative.

COMMENTS AND OPERATION

This is a fairly typical history in every way. It is typical in the fact that the man had an injury; that he has had a depressing injury, which is not always the case; that he had a paralysis of speech and paralysis of his right hand and in part of his right leg. This has materially improved. His speech has materially improved, but his right hand is still somewhat incapacitated. It is the center of disturbance.

The immediate effect of injuries of this class is recovered from, as a rule. Handled in anything like a careful aseptic manner, even with drainage, they recover, and they recover without drainage. But the immediate recovery is only a part of that which is desired in these cases. Next comes the restoration of function in the impaired convolutions of the brain, and we know that we have many associated centers; that a center may cease to functionate for a short time, or even be removed, as I have seen a spoonful of brain tissue removed, and the function of the convolution, as we understand it from a physiologic standpoint, be eventually restored either completely or partially.

But that is not all. The important thing in every head injury, whether it is a compound fracture or a simple fracture, or whether it is a concussion of sufficient severity to produce unconsciousness, all centers around the fact whether the patient is subsequently going to have epilepsy, or whether he is subsequently going to have these epileptic convulsions and finally become a victim for the asylum. That is the important point.

The injury received does not need to be a hammer-blow: it can be any blow on the head that produces unconsciousness. For a long time we believed that the Jacksonian type of epilepsy was the sequence of a compound fracture, and rarely ever the sequence of a concussion. We are now learning that a concussion very frequently results many years later in epilepsy, and that led the late Dr. Fenger, who was the father of it, to adopt a treatment which he felt would obviate the ultimate epileptic convulsion.

There is another lesion which is a sequence of trauma not associated with fracture, frequently not even associated with unconsciousness, and that is an epidural or subdural hemorrhage, with the formation of an encapsulated cystic condition, either outside or inside the dura. This gradually produces an increasing effusion and increasing irritation, and finally the patient commences to show, if not convulsions, at least changes in his mental condition, and he finally lands in an asylum, never having had a depressed fracture or anything more than a contusion or a concussion. In some of these cases you have momentary stunning of the patient. The patient's scalp is most always not cut. He does not have the period of reaction immediately after the injury and subsequent unconsciousness that one has in the middle meningeal hemorrhages. But he does have a circumscribed cystic formation, hemorrhagic, we believe, frequently later of a serous or grumous character, with a mass of connective tissue around it, slowly increasing in size, and, as I have said, the patient winds up in an asylum. Preceding the disturbance of his mind he has repeated attacks of headache, these attacks increasing in severity. That, too, is very common with a traumatic epileptic patient. The period of time that may elapse between the injury, from our personal observation, where the injury is clearly the etiologic factor, ranges from a few weeks to over thirty years. In one patient it ranged over thirty years, in another, twenty-seven, in another, twenty-six, and in many, twenty-five and twenty-four years, but the greater number appear under twenty years after the accident. That has been one of the important factors in giving a certificate to an individual who has received an injury to the head sufficient to make him unconscious. To show you how much stress I place on that I have often said that rather than be rendered unconscious by a blow on the head I would prefer to die—so much fear have I of the secondary lesions that occur in connection with these injuries.

This man had a depressed fracture, as you see, as it stands out when he is under the anesthetic. Before he was put under the anesthetic there was a pronounced stellate depression, the same as you see it here.

In the management of a case like this we usually make a U-shaped flap wide of the lesion, and expose the margin of the bone. I am not going to take out a bone-flap, but make a subperiosteal removal of the bone. You see the margin of the bone. I will enlarge the margin as much as I think it should be enlarged, but I will probably remove a considerable portion of the dura.

The question comes up in all these cases as to why we have the epileptic convulsions. Are they due to the irritation of the dura, or are they due to irritation of the brain. That has not been definitely settled, but it is my conviction, from clinical observation and from some experiments made many years ago, that it is the irritation of the dura that causes the convulsions. I remember a case that occurred when I was a student in Rush Medical College, at which time a celebrated French physiologist made a demonstration of convulsions occurring in a dove from injuries to the dura. Now, the dura will be adherent to this. I want to preserve my skin-flap. I want to keep within the proper zone. I am deep down on the dural tissue, because subsequently I shall remove a portion of tissue that rests immediately beneath this scar, because it is my belief that the convulsion is due rather to the cicatricial tissue and irritation of the dura than to the irritation directly of the brain tissue itself. There is the cerebrospinal fluid, as you see, and here is the dura. I shall lift that up. The dura is involved in this large mass of cicatricial tissue. There, I believe, is the cause of the convulsions. It is the irritation here which I believe produces the convulsions. Now I am going to get under the adherent margin of bone. This attachment shows how little of the osteogenetic force we have in the internal layer of the periosteum, that is, in the predural periosteum. It shows that we have but a few of these feeble osteogenetic cells, differing from the osteogenetic force of the periosteum in other positions of the body. You will see, it has not attempted to span this gap; that is, it does not build bone, whereas in the tibia and femur many times you will find it spans a gap an inch and a half and even two inches, frequently an inch, and very commonly smaller distances. I am turning down the external periosteum just above the beveled zone, so as to get under the predural periosteum without any danger or difficulty. As soon as I have this flap raised from the margin I will have no difficulty in separating it for a greater distance. The only bleeding we will have will be from the veins of Santorini, which one encounters in doing the Gasserian operation.

I have detached the dura from its fixed position all the way around. There has been no tendency to hardening here. There has been a depression that we believe is due to the contraction of the tissue. Now comes the important factor. In our management of these cases we have found that with the removal of this irritating zone of the dura we obtain the best results that can be obtained by any line of treatment, not merely detaching the dura from the bone, but excising it, and then we will interpose a thin plate of paraffin, preventing adhesions from forming. This coal-tar paraffin is non-absorbable and non-irritating, and remains permanently of the same size and shape, differing from the whale-oil paraffin, which is a normal product and absorbable. The coal-oil paraffin becomes encapsulated with a thin layer of connective tissue. I go clear out to the margin of cicatricial tissue, and free it for a little distance back on either side. You see how dense and firm that is from the pressure which is required to cut it with this high leverage scissors. There is the central cicatricial mass, and here is the connective tissue beneath it. There is the cortical substance. We have taken out all the cicatricial portion of dura, and in removing that we have removed the great source of danger. This man is at an advantage in being operated on at this time, namely, he has only had two convulsions. He has not had the epileptic habit established. In the second place, he has the advantage in that the force of the blow was concentrated over a small area, differing entirely from a man who has a fracture of the skull with the force of the blow distributed over a very large area. I force this paraffin in underneath the skull on either side. That protects the brain from attachment to the dura or scalp, all over the injured zone.

In some operations that have been suggested in cases of this kind a considerable portion of the cortex has been removed. The cortex comes up clear, not cicatricial, perfectly soft, and the removal of that would be detrimental and would add nothing to the percentage of cures of the epileptic attack. We have had lots of experience with paraffin, but not so much in putting it in as in taking it out, particularly when operating for hernias that have had paraffin injected, and we have had to remove the paraffin subsequently because it did not cure the hernia, and we know what a thin layer of connective tissue was deposited around it.

In estimating the treatment for ankylosis we have figured a number of times that if we could get a material like paraffin that would not be absorbed, and, at the same time, that would not become vascularized or infiltrated with connective tissue, and would not fracture from pressure, we would have an ideal interposing material in many of the joints, if it would stand pressure. The trouble with paraffin is it does not stand pressure. If you put pressure on it and it is cold, it will break. If you put pressure on it when it is warm, it will press out. It would be desirable if we could find material that would not do this. To remold it down into conformation is not a difficult proposition. The interposition of the flap and retention of the flap, with its vitality, is a difficult proposition, and adds most to the obstacles we have in getting good results in these cases of ankylosis, because of the quantity of connective tissue that forms around the new flap and the joint where it was placed.

We never drain these wounds. When we operate for a trifacial lesion, where we have such a large quantity of blood escaping into the cranium, when one is closing it, in fact, when there is a blood-clot an inch in thickness almost, one has no fear at all, but let it form, it is rapidly absorbed, and it does no harm.

Let the record show there was a cicatricial mass one-eighth inch in thickness in some of its area, covering over the exposed portion of the dura; that it involved the pia, and in some places the convolutions; that this was freed first from the scalp and from its bony margin over one-half inch all the way round. It was then excised on a line with the bony margin, taking off just the surface of the convolution. In the second place, sutures were used to stop the bleeding, which was very slight during any part of the operation. A plate of paraffin one-tenth inch in thickness was then inserted on the surface of the brain and under the bony margin. It was adjusted to give it pliability. It was gradually worked under the margin of the bone, so that it separated the dural margin from the bone and was elevated in the center to the level of the normal bony elevation, and that it was a little lower under the margin. When this was inserted, there was no bleeding. Let the records show that the dura could not become adherent to the lacerated or fractured edge of the bone, and that there was no opportunity for the dura to become subsequently attached to the scalp.

May 10th.—This man's speech is improved 70 per cent. over what it was. He can use his arm much better, and has resumed his occupation as a coachman. He has had no headaches or convulsions. His employer, a physician, believes that with his present rate of progress the patient will be completely restored in three months.

TRANSPLANTATION OF BONE

Osteitis Fibrosa Cystica

HISTORY

(Written June, 1911)

B. K., male, aged eleven. Family history negative. Had measles; otherwise always well.

Present Illness.—In September, 1908, he was run over by a bob-sled, the hind runner running over his right tibia at the middle of shaft. Had severe pain and swelling over that area. There were no fractures. Patient remained in bed for one week, when the pain disappeared, but some swelling remained. One year ago his mother noticed that the swelling was gradually increasing in size, but the patient did not complain of any symptoms; no pain, no chills, and no fever, and he did not favor the injured leg.

Four weeks ago patient slipped on a stone curbing and struck the same area of right tibia on the curbing. He had severe pain in bone and swelling rapidly increased in size. A week later he was operated on. The bone was cureted and a part removed. Mother took a specimen of bone to a Chicago hospital, where she was told it was a spindle-cell sarcoma. After the operation, three weeks ago, patient made a rapid recovery, but swelling still remains. There is also slight pain. No chills or fever. No loss of weight. No night-sweats and appetite good.

First Portion of Operation by Dr. Golden.—An incision was made over the left tibia, a portion of the tibia 9½ inches long by 1 inch by ¾ inch was removed from the crest. The periosteum was removed from this bone. The bone was laid aside, being very careful about handling it. The periosteum was sutured with catgut over the 9½-inch trough, where the bone was removed and the skin closed with horsehair. A collodion dressing was applied.

Second Portion of Operation.—An incision was then made over the right tibia, near the tumor mass. Eight and one-half inches of the tibia was removed with the tumor. Each end of the bone, upper and lower, was notched out, and grooves were made in which to place the ends of the bone to be transplanted. The sharp and roughened edges of the distal and proximal ends of right tibia were removed with bone-cutting forceps. The portion of bone 9½ inches to be transplanted was then placed in the gap in the right tibia, its ends being placed in the holes made in each free end of the right tibia fragments. Measurements were carefully taken, so as not to get any shortening, and two nails were then put through the proximal and distal ends of the transplant and tibia to keep it from slipping into the tibia too far. Muscles and fascia were then sutured over the bone with fine wire. Skin closed; collodion dressings put on. Each leg was put into a plaster cast extending from the toes to about the knee-joints.

OPERATION BY DR. JOHN F. GOLDEN; COMMENTS BY DR. JOHN B. MURPHY

Dr. Murphy: How long after the trauma before the swelling was noticed?

The Intern: One year.

Dr. Murphy: How do you figure that? The accident occurred in 1908, and you say one year ago, and it is four years since 1908. How do you figure that? What has become of the other years?

The Intern: This history was written two years ago, Dr. Murphy.

Dr. Murphy: All right; that clears it up. Go on with the history, doctor.

Dr. Murphy: When was the first operation done?

The Intern: In July, 1911, by Dr. Golden.

Dr. Murphy: Yes, that is right, Dr. Golden did the operation at that time. He took out all the bone in which the cyst was situated. How long was the transplant?

The Intern: Eight inches.

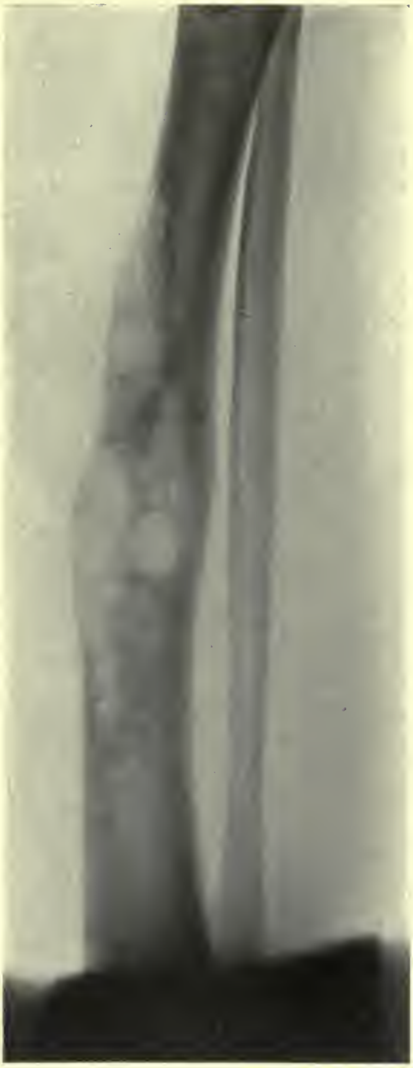

Fig. 114.—Skiagram showing tumor of tibia before first operation.

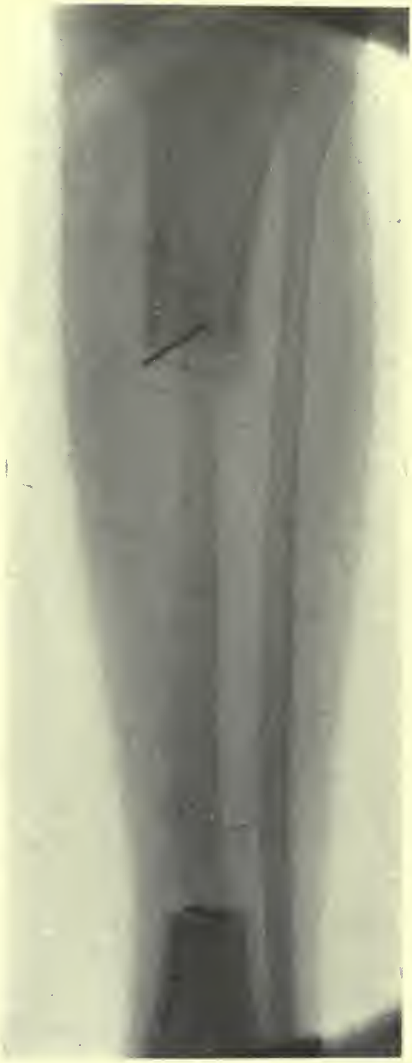

Fig. 115.—Skiagram showing transplant after first operation.

Dr. Murphy: Eight inches? Is that right? It seemed to me it was 9 inches or 8½—which was it, Dr. Golden?

Dr. Golden: Nine inches and a half.

Dr. Murphy: That is an illustration of one of the cases in which we have done this bone transplant work. Dr. Golden did this work during my illness a year ago and he succeeded in getting a full growth of bone, except at the lower end, where the nail was put in. The skiagram shows that there was a little suppuration at that point, and that there was a little necrosis and absorption. It was not of the extreme suppurating type. This bone has regenerated beautifully.

There is the splendid result Dr. Golden got with a transplant 9½ inches long placed into the space from which he removed the diseased portion, which was diagnosed first by one pathologist as a spindle-cell sarcoma and by a second pathologist, subsequently, when the whole mass was taken out, as osteitis fibrosa cystica. That just shows us how significant the microscopic diagnosis may be, or, rather, may not be. We have come into the habit of accepting that as final, but it is not final at all.

From the gross appearance of the skiagram before the operation one would conclude that it looks more like an osteitis fibrosa cystica than like a sarcoma, and that picture is more valuable than any picture that the microscope shows, because sarcoma of the spindle-cell variety, of the large round-cell variety, and of the small round-cell variety destroys bone in a definite way. The chondrosarcomata destroy bone in a definite way, so that you can draw your conclusions just as well from the skiagram as from the microscopic picture, and you can form a fairly accurate estimate before you start your operation and before you remove a portion of that bone as to the line of procedure you are to follow.

If this diagnosis of round-cell sarcoma is correct, then amputation is the only procedure that should be done at this time, so far as we know; if, on the one hand, it is a large spindle-cell sarcoma or a giant-cell sarcoma, excision will probably give a final cure. If it is a chondrosarcoma, there is about an even chance of removing the bone and getting a final cure. A chondrosarcoma will recur as a metastatic sarcoma in some other position in the body. Therefore, the conservation of the limb, instead of amputation, is the procedure that should be attempted in all these cases, and that has led to the large number of cases we have had of removal of great areas of bone, and to a complete and perfect reproduction of bone following transplantation.

In Dr. Golden's case there is complete healing in of the lower end of that bone, but a non-union. This boy must have a solid bone from one end to the other. He has already given up a portion of the bone on the other side once. We will take out another piece, because bone reproduces itself all the time. I would suggest taking out another piece at the lower angle, but before we take that out we will expose this tibia and see how the end of the bone is. This is our first opportunity of getting a look at a bone that has been transplanted, because all the other cases have healed, and all have gone to a good and perfect result and there was no excuse to take out the bone. So Dr. Golden will expose the tibia and put in a new transplant, inserting the upper end into the first transplant and the lower end into the tibia.

In making that flap Dr. Golden remembered that primary union of the soft parts is absolutely essential. If you get infection, even of the least virulence, you lose your entire sequestrum. He will not lose it in this case, however, because it is already filled in with new bone. Now, then, the doctor is managing to get his flap out here, keeping on the inner margin of the tibialis anticus, exposing the lower end of the fragment, pulling it up into position, and then exposing the other.

I want to call your attention to the points I made as to diagnosis, because this helps every man. The general practitioner can see it as well as the microscopist, and draw his conclusions from the skiagram. The lobulated condition of that bone is very evident. It is different from the destruction produced by sarcoma of the spindle-cell type.

These bone transplantations have to be done on definite principles, and the definite principles concerning bone transplantation consist of—(*a*) The fragment must be taken out with

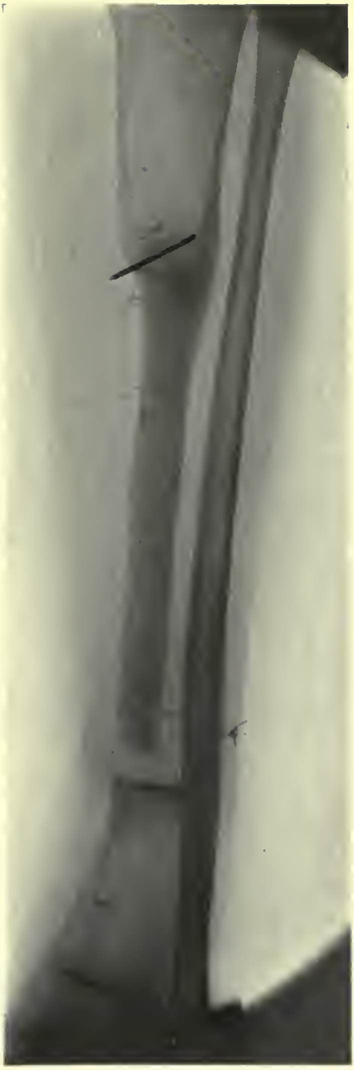

Fig. 116.—Skiagram before second operation, showing disappearance of lower end of transplant, due to infection setting in three weeks after first operation.

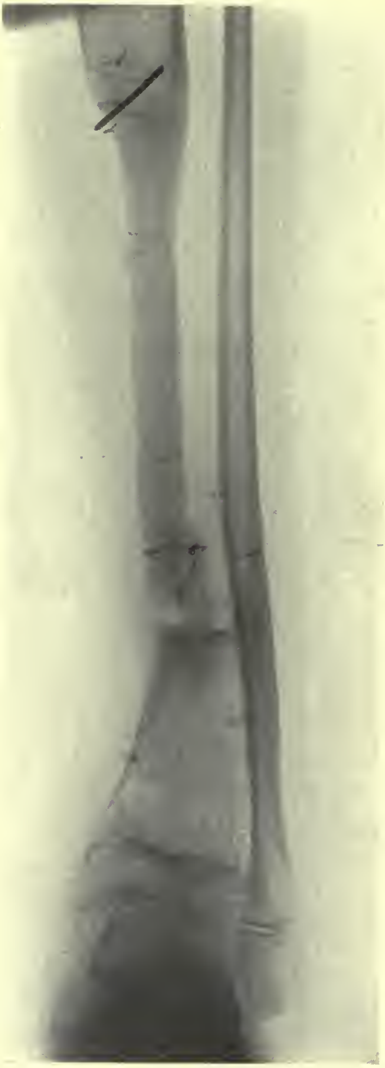

Fig. 117.—Skiagram showing second transplant in place.

or without the periosteum; (*b*) preferably from the same patient; (*c*) if it cannot be obtained from the same patient, it should be obtained, as near as possible, from a patient of the same age; (*d*) when transplanted, it must contact at one or both ends with living healthy bone. It reproduces best when it contacts at both ends. It is better to insert it into the medulla at both ends, but that is not at all necessary. The rôle which the transplant plays is merely that of a scaffold. It always dies. The Haversian canals in the dead bone act as tubes into which the Haversian vessels from the living bone above and below pass. These Haversian vessels from above and below carry with them on their walls osteoblasts and osteoclasts. The osteoclasts dissolve the bone that was transplanted, and the osteoblasts make new bone, so that one brick is taken out and another brick is put in, and long before—months before—all of the transplant is removed you have new tubules in all the old tubules, and firm bony union of the living and dead. There is the interesting thing about it. That continues until all the old bone is removed. New lamellæ are produced around it, and it is all new bone. Then the new bone increases in size and strength as much as is necessary or demanded of it by the extremity.

In a case of osteomyelitis of the tibia in a little girl about eight years of age, the transplant contacted with two little spiculæ of living bone at both ends. The piece we put in measured 5½ by ⅜ by ⅝ by ½ inch. That operation was done in 1908. I saw this patient within the last two weeks. Her leg has grown; she is not in the least lame, and does not know that anything has been done to her leg at all, showing how perfectly the bone can be reproduced. In that case we had to transplant the fibula into the epiphysis of the tibia to prevent shortening of the leg during the period that suppuration was going on. Remember you can never insert a transplant in the presence of pus. Wait until it has entirely healed. That is why Dr. Golden waited so long in this particular case. He waited until it had entirely healed before he put in the additional piece of bone to overcome this separation. It has formed an arthrosis, developing a nice joint. Here is a beautiful joint produced by nature when we did not desire it, and we took advantage of that knowledge in our arthroplasties. We make new joints readily where the bones of the joint have become ankylosed.

In this case the periosteum is united to the upper fragment. Dr. Golden is peeling off the periosteum from the upper side and he is going to expose the medulla on the lower fragment and take out the fibrous tissue that has formed between the lower and upper fragment, so as to expose the medulla of the lower fragment. Look at this bone—the transplant! See how perfectly the periosteum has grown right to it. Dr. Golden will transplant the piece of bone that he will take from the opposite tibia onto the surface. That shows how nature fills up the gap long before it shows in the skiagram. The skiagram does not show that it is all filled up with new bone, but the gross appearance shows it very well.

Now Dr. Golden is reaming out the medulla in the lower fragment to get room to drive in the transplant. He presses the lower fragment down and the upper fragment up. He transplanted the first piece of bone without the periosteum and it has all reproduced. It is reproduced by Haversian vessels that have gone in from above. This bone will enlarge so that it will be as large as a normal tibia.

Another case we had, similar to this one, except that the disease involved the upper end of the humerus, shows what a detriment the periosteum is to repair. No bone was produced on the white line seen in the skiagram. That is the periosteum which we did not remove from the transplant. Many months afterward the white line is still to be seen, showing where the periosteum interfered with the reproduction of bone. The periosteum is not necessary at all. In analyzing the reproduction and growth of bone I have used this simile in my article in the "Journal of the American Medical Association," namely, that the osteogenetic force is represented by a tree. The trunk of the tree is the medullary canal, which is the one place that retains osteoblasts in the greatest uniformity throughout the entire period of life. The first bifurcations of that tree are the Haversian canals, and next are the canaliculi and lacunæ, and finally the leaves are represented by the periosteum. Every one of these spaces contains osteoblasts, and every vessel that goes through the medulla carries with it osteoblasts, and they fill up all the transplant with new osteoblasts—first vessels and then osteoblasts—and produce new periosteum with osteogenetic cells.

Here in this clinic we apply our knowledge of the regeneration of bone to five different pathologic conditions: First, in making up for congenital defects, as a flattening of the nose, absence of development of the mandible, failure of development of the radius and ulna and tibia and fibula—never or rarely of the humerus and femur. I have named these aplasic osteal conditions in the order of their frequency. The most common is the nose. In order to fill these spaces you can transplant a bone from any other position in that patient's body that you desire. In making that transplant, whether it is in the nose, in the mandible, in the femur, in the tibia, or in the humerus, you must put the piece that you take from another position in the patient's body in contact with living bone where you implant it. Is that clear? In other words, if you take a piece of bone, with or without its periosteum, transplant it, and, except in very young children, it always dies and fails to reproduce bone and entirely disappears. I transplanted a thumb eight or ten years ago in a case of double thumb; took off the skin and underlying tissues, and put the thumb in to fill a defect in the nose. It healed perfectly and I got very much elated over my success. But in a year and a half the whole thing had disappeared, because I did not take off the periosteum from the transplant, and because I did not contact the bone which I transplanted with living bone denuded of its periosteum, so that the Haversian vessels could get from the living bone into the transplant. If you have that clear you have the whole key to success in bone work, except the mechanical difficulties and the element of asepsis. In doing that work remember that asepsis is absolutely necessary, otherwise a failure occurs. Remember that mechanical exactness of application of bone is necessary, and when you are doing arthroplastic work do not try to do it after reading my article in the "Journal of the American Medical Association." Read over the article if you wish, and go to the dead-house and do a half-dozen or a dozen operations on the cadaver. In doing that many you will be doing just a tenth or a twentieth of what I did, and then read over my article again and again, and if possible go and see some one do these operations.

I just had the painful experience, within the last forty-eight hours, of telling a very able man that the joint which he made was just as surely going to be a failure as the sun was going to rise, because he did not recognize some of the essential things in doing the work, and Murphy's method will be blamed for the evil results.

Arthroplasties are not easy to do. They are difficult to do; they are not difficult if a man exercises the average intelligence which a cabinet-maker uses or a shoemaker has to exercise before he patches a shoe. A doctor does not possess any superior power, in a mechanical way, to attempt to do mechanical things without preparation more than people do in other walks of life. You can do it. Every doctor should do it. He should give his careful attention to the preparation. I say this today, I think, for the first time, on account of the painful experience I had with an able man in the last forty-eight hours, showing how essential it is to recognize things in detail to get results. Every one who desires to do this work can perfect himself to do it as well or better than I can do it, because it is not difficult if he is prepared for it in a regular and systematic way. It is difficult if you have done no cadaver work and start in by making new joints in the living. You meet obstacles which you will not overcome or even recognize until six weeks after the operation. Then you have spoiled the knee or other joint, and spoiled it for anybody else doing the work.

I want to call your attention to another matter, and that is how perfectly this bone has been reproduced. I want you to look at the thickened new periosteum; how the periosteum has thickened and filled up after the other bone was removed, and how much more vascular it is than the normal periosteum.

That was point No. 1—the treatment of aplasic conditions. Point No. 2 is the good use to which bone transplantation can be put in securing the reunion of ununited fractures. It is the best and only regular medium of getting union. The mere freshening of the bone following ununited fractures fails in a large percentage of cases.

We had one such case where a Lane plate was used to maintain fixation of an ununited fracture of the humerus. Nothing could be more beautiful than the apposition of the ends of the fragment, and not the slightest effort at the production of bone is evident—a non-union with a most perfect apposition, and that is the most frequent position of non-union in the human anatomy.

In the treatment of ununited fractures, as illustrated in this case, the first thing is to remove the foreign body and clean out the plastic focus and get complete union without infection; then freshen the ends and drill a hole into each one. I have an instrument made especially for this purpose—a medulla drill or reamer. Drill a hole in the medulla and enlarge it to any degree you desire. Then take out the transplant from the crest of the tibia, with or without the periosteum, and drive the transplant into each fragment. Here the transplant was driven down into the lower fragment an inch. Then take a rectangular forceps and drive the transplant into the upper fragment as far as you wish.

In one of these cases we made three skiagrams. Two showed a fairly good union. One showed that there was no union at all, showing the significance of taking pictures at two, three, or four different angles so as to be sure you have all the lines of fracture in the *x*-ray field. Why bone is not reproduced we do not know; whether it is because the fragments are too perfectly immobilized we do not know. You must always take into consideration the class of patients you are dealing with. At the Alexian Brothers Hospital we got the lower class of people, who were overworked and underfed, and it was common to have non-union. The patients at the Cook County Hospital are usually underworked and overfed, and there we very rarely had non-union; but the man who works hard to support his family is always underfed, overstrained, and overworked.

The next class of cases in which you utilize your knowledge of replacing bone that is dead is in fractures of the head of the femur and head of the humerus. We had one case of fracture of the head of the femur. There was rotation of the fractured neck or head of the femur. The head died. We did not see that case until seven weeks after the accident. The head had been dead all that time. We rotated that head around and nailed it to the neck of the bone. Only a little of the head was absorbed. We saw this boy the other day—a week ago last Sunday—and had him walk up and down the corridor before the new interns, and they broke about even at guessing which was the leg operated on three years before. The last skiagram taken just a few weeks ago shows how the epiphysis continues to grow and the nails push further from the joint. The original operation was done in October, 1908. That shows how perfectly that leg has developed, and how that chap has developed into an active young man without a limp.

In a case of fracture and luxation of the head of the humerus we had to take out the head first and detach it. We freshened the distal end of it and then freshened the proximal end of the distal fragment, replaced the head in the glenoid cavity, and nailed it on the distal fragment. All that transplanted head has acted as a carrier for Haversian vessels and reproduced new bone of exactly the same size and shape as the older bone.

The next condition in which we utilize our knowledge of the transplantation of bone is for the replacement of malignant disease or non-malignant disease, as chondrosarcomata. A young girl, about sixteen years of age, had a chondrosarcoma of the upper end of the tibia, almost involving the joint. We took it out, leaving the articular surface of the tibia intact. Then we put in a piece of tibia taken from the opposite leg, and this girl is back on her feet and is having a reproduction of bone of the regular size.

A man had a traumatic chondrosarcoma of his trochanter. We removed the upper end of the femur entirely and implanted a piece of bone from the opposite tibia, suturing the upper end into the acetabulum. Reproduction of bone progressed rapidly and he has a useful leg.

We never take out the nails which we put in. If you get suppuration or anything around the nail in the process of repair, even pyocyaneus-infection, then you take that nail out, but the great bulk of our patients go away with the nails in and never come back to have them taken out.

There is no question as to what will happen in this case. If it is aseptic, and he has a contacting of the fragment with living bone, only one thing can happen, and that is filling of that fragment with osteoblasts from below, if not from above, with a reproduction of bone from both sides. This will give a good result in this way. The wire which we use in these cases is made of 12 strands of phosphor-bronze, just as thin as the thinnest silk thread.

The operation will be completed in the usual manner, closing the wound with catgut in its deeper layers and horsehair on the surface. A plaster-of-Paris cast, cut with a Gigli saw after it has hardened, will be put on and kept on for four or five weeks. The boy will not be permitted to put his weight on this leg for several months until we are convinced that the bone has regenerated completely.

CARCINOMA OF THE LIP

HISTORY

Male, aged forty-two, admitted to Mercy Hospital May 13, 1912. Came to hospital because of a growth on lower lip. Family history negative.

Present History.—In October, 1910, patient noticed a lesion resembling a cold-sore on the lower lip, to the left of the median line. It was eroded and raw, and was not benefited by local treatment. He never traumatized the lip, nor has he smoked a pipe for the past twelve years. About four weeks after its appearance the growth was slightly elevated and hard. It increased in size slowly. Two months after the appearance of the growth the patient had two treatments with the electric cautery, two weeks apart. Following this treatment the mass seemed to disappear for three months, when it again appeared and increased slowly in size.

In July, 1911, he had the lesion cauterized again, this time destroying quite a portion of the lip and leaving a cavity. The growth recurred again. A plaster was applied and this was followed by a slough. He later had two more applications of paste. Since the paste treatment the mass has not come above the surface level, but it seems to be infiltrating the surrounding tissues. For two months past there has been a burning, drawing sensation in the old scar, and there is a slight glandular enlargement on the right side. No loss of weight. General condition good.

COMMENTS AND OPERATION

Dr. Murphy: How did it appear, doctor? Was it first a sore the size of a pea, or was it first a scaly lesion with a scab or crust formation? Did that crust fall off from time to time? Did it enlarge? How did it appear?

The Intern: The patient described it as having a scaly appearance.

Dr. Murphy: You gave the patient's description. What a difference there is between the doctor's description and the real facts in the case. To get some idea of what the lesion was primarily one must have an accurate description of it, and an accurate description is lacking.

With the exception of the description of the scar, that is about the clinical course of these cases, but that is just what the clinical course in these cases should not be. In every article that one reads on the subject of cancer, epithelioma, or adenoma this statement appears: "If we could get these cases earlier, we would have better results." We are not getting them earlier; in fact, I think we are getting them later.

In cancer the question that first arises—when one analyzes the statistics, the clinical course, and the final outcome of the case—is, how soon after the first erratic epithelial cell penetrates its basement-membrane, which is the one condition that makes it a cancer—how soon after that do these cells pass into the neighboring lymph-spaces, and how soon are they passed on into the first lymph-filter, the first lymph-node? Up to the present time that question remains unanswered; we have no light on it. There is possibly a definite reason for that. Experimental work with mouse cancer has shown that it does not metastasize so frequently as does cancer in the human being. We are not justified in saying that if we had the cases earlier we could get results until we know something about the transmission of the cancer-cell from the primary penetration of the basement-membrane to the nearest lymph-filter.

In a recent article in one of the British medical journals there appeared an analysis of cases of epithelioma of the lip extending over a period of twenty or twenty-five years, and the author endeavored to establish the final outcome of that class of cases. I must say that when I read that article I received a great shock. It was demonstrated, from the cases that could be traced, that 52 per cent. of the patients who had carcinoma or epithelioma of the lip without any demonstrable metastasis at the time of the operation died a cancer death! In the case before us today the carcinoma was visible from its very onset,—readily seen by physician and layman,—and yet what has been done to lower a 52 per cent. death-rate from cancer?

What is "early" intervention in this class of cases? Cautery and paste! When these cancers are in some hidden zone where we are unable to demonstrate them by symptoms or physical signs until they have developed to a great size, there is some excuse for failure to operate and we know what we can expect as a mortality in that class of cases. But, compared with the cancers that are so easily recognized and yet give so frightful a mortality, why should there be a difference?

There is a difference,—a well-recognized difference,—and that is the redeeming feature. It is based on anatomic reasons. The hidden ones are later recognized, when the lymph-supply of the zone infected by the carcinoma shows evidence of involvement. A carcinoma is or is not dangerous, from a metastatic standpoint, in proportion to the lymph-supply of the zone primarily involved. Carcinoma occurs more frequently in the rich lymph-supply zone than in the lesser lymph-supply zone. In other words, carcinoma occurs always near the active lymph-zone of a viscus, as in the pylorus, the cervix of the uterus, the cervix of the gall-bladder, and so on. Carcinoma rarely occurs in the fundus of the stomach, the fundus of the uterus, and the fundus of the gall-bladder. The richest lymph-supply in all these viscera is found in the cervical portion of each viscus. Carcinoma of the fundus of the uterus metastasizes very late, and frequently does not metastasize at all. If you analyze the statistics of cancer of the fundus of the stomach, you will find that it gives the best final result. Carcinoma of the lesser lymph-supply zones of the large intestine gives the best final results, while carcinomata that occur near rich lymph-supply zones of the intestines, as well as in the cervices of the organs mentioned, give bad final results.

Now let us come back to the lip. The lip is one of the richly supplied lymph-zones. Metastasis should and does take place earlier here than it does in any other position of the body.

How long after the metastasis takes place is it before the carcinoma shows itself? In analyzing carcinoma of the lip we find that there are two distinct types of return: one occurs in the lip itself, in the lymphatics, far from the point of primary invasion, and the other occurs, first, in the lymphatics of the upper triangle of the neck; second, in the deeper structures of the neck, and, third, one commonly overlooked, in the small lymphatics situated just below the foramen mentum. This zone is overlooked in making the examination practically all the time.

In the case of an epithelioma that we consider to be one of the most innocent types of cancerous tumor, the mortality is 52 per cent. where there is no demonstrable metastasis at the time of the primary operation. Added to that frightful mortality is another 24 per cent. of deaths occurring in those cases in which there is no demonstrable enlargement of the gland at the time of the operation. In other words, 76 per cent. of the patients who have a demonstrable enlargement of a gland at the time of operation die a cancer death. Were it not for the difference in the lymph-supply that statement would be so appalling that we would not be looking for the removal of a carcinoma of the pylorus or a carcinoma of the intestine, but the later transmission and the clinical fact that some patients remain permanently cured—true, the number is small—make us still hope to get some results from surgery even in the deeply situated carcinomata.

In the analysis of the next greatest zone of frequency of carcinoma, that of the mammary gland, we find about an equally unpleasant situation. The improved technic that has been adopted in recent years,—first, that of Halsted; second, the splendid technic of Rodman; and, third, the additional advance brought out by Handley,—would tend to make us believe that we could get better final results in the future than we have been having in the past. That appeared to be true for a long time until Halsted analyzed his statistics at the meeting of the American Surgical Association in Cleveland. That analysis certainly did not justify us in cherishing much hope.

In the case before us the scar is the paste scar. On one side there is a little more invasion than on the other. How much of this is scar and how much is carcinoma we do not know. We have not removed a portion of it. The growth has returned after removal by cautery and paste and is becoming thicker in the middle. We will excise this entire mass, making a V-shaped incision, one limb of the V extending from the angle of the mouth downward and inward to the base of the lip, the other limb extending from about the center of the lip downward and outward to meet the other limb of the V. None of the blood has been admitted into the mouth. We have packed behind the lip with gauze. The ether vapor passes in through a rubber tube clamped into one nostril, so that no blood can pass down into the lungs or stomach. Having excised the tumor-bearing tissue, we will begin our dissection, searching for metastases in lymph-nodes.

We go, first, into the most dangerous zone for the situation of a secondary node, just over the mental foramen, which I can palpate. That node is recognizable, as a rule, by palpation, and we can remove the node and the underlying periosteum. There is some bleeding from the artery in the foramen. I free the lip-flap from the chin, so as to permit it to slide upward to fill in the gap left after the removal of the tumor, when we are ready for the repair. There has been an infiltration here from the necrosis produced by the paste. We can now inspect this zone of the foramen, and I will remove the connective tissue and periosteum down to the margin of the opening, so that there is no chance of leaving the lymph-node. We will carefully examine that tissue to make sure that it is free from carcinoma.

Now we will make a section of the lip, divide it upward and backward so as to make a new lip, and then slide it forward. I am lengthening the internal incision so as to free the mucosa and thus secure more mucosa for the new lip. That is very important. The new lip must be lined with mucosa throughout its entire extent, and skin and mucosa must be approximated accurately. If we do not have ample mucosa, then we have the scar fixed to the bone and there is a tendency to continued irritation and subsequent immobilization of the lip. In the plan of the operation of reconstruction of the lip my attention is devoted to the mucosa primarily because, from the patient's standpoint, that is very much more important than the skin.

The next zone of danger is the lymph-node that is situated in the anterior pole of the submaxillary salivary gland. This node is situated *in* the pole, not *on* the pole. The mouth is closed so there is no bleeding into it, and we are ready to remove the submaxillary lymph-node. That node is scarcely palpable in this case. We know where it is because it is always in relationship to the salivary gland. It is situated in the anterior pole and just a little to the inner side. I am endeavoring to locate it. I can feel it, and, as I pick it up, I can feel that it is resistant to pressure. We will take off a portion of the under part of the salivary gland and with it we will remove that lymph-node. It is separated from the rest of the structure. I have taken it right out of the capsule, and it stands alone and independent of the gland-structure after the capsule is divided. That is the point in which we see the most frequent recurrence of carcinoma of the lip.

How far should we go in the removal of these glands? How far should we go in the removal of the tissues? When you take off a large piece and you succeed, you think you did wisely and well; when you take off a small piece and succeed, you think you did as well; when you take off a large piece and fail, you conclude you did not do well, regardless of the size of the piece that is removed. I think one can say that of carcinoma in every position in the body. How far one should go in the removal of these glands is illustrated by the work of Wertheim, who has had such brilliant results up to date. But remember, that is only *up to date*. Let us wait until another decade has passed; let us wait until the same period of time has elapsed as has elapsed since the Halsted operation was first done, and see where we will arrive, so far as recurrences are concerned.

In the article by Halsted to which I referred a moment ago, there are reported either 111 or 211—111, if I remember correctly—cases, and in 61 of these there were no demonstrable metastases, either macroscopically or microscopically. Deduct these 61 cases, and what was the percentage of mortality in the remaining cases? In other words, what has been added to our results over those which our forefathers obtained when they merely removed the breast? We must deduct the cases with demonstrable metastasis, because we all know of the splendid work done at Hopkins, and how carefully they examine their specimens. If there were any metastases, in all probability they would have been found.

An analysis of that class of cases disheartened me very much, and an analysis of the more extensive operations done in various portions of the body later has added to my discomfort, and has made the cancer field rather a hopeless proposition to me. There has not been any material improvement in a quarter of a century. I think we can honestly say we have not improved our cancer results in the last quarter of a century.

We will next direct our attention to the anterior angle of the skin-flap. It falls into position easily because I freed both the right and left flaps. I will put in a deep suture as a tension suture, merely to swing that flap into its proper relationship and hold it there without pressure.

We are now ready to proceed with the construction of the angle of the mouth—a very important step. I am shortening the mucosa and the skin-flap of the upper lip, so as to have the mouth open a little farther to the side and give an ample opening, and that is the all-important thing. The redundant skin and mucosa will pull back to position. The same plan is used on the inner side for the approximation of the mucosa far back, so as to keep an ectropium of the lip from occurring. The upper approximation of the mucosa to the lip is done with silkworm-gut. We are getting an ectropium of the mucosa; that is, it is in excess on account of the method in which the flap was prepared. The angle of the mouth is in this manner reconstructed, so that it is practically the normal angle. The external skin-flaps are then united, and the operation is completed. While it is important to remove enough tissue, it is equally important to secure flaps which will permit of accurate approximation without tension or a minimum degree of tension.

How long after metastasis occurs it is before it shows itself in the enlargement of the gland in epithelioma of the lip we do not know. That is one of the things that originally called our attention to the question of how early it does take place. We had in this hospital a patient we operated on for epithelioma of the lip. The man came back nine years afterward with an enlarged lymph-node—it was in the time preceding the removal of this node—situated under the anterior portion of the submaxillary salivary gland. The question came up, How long had that infection been present in that gland? It had not returned *in loco;* but nine years after the original operation we removed a squamous-cell epithelial growth from this lymph-node, which means, what? That it had been there nine years, because there is no such thing as a primary squamous-cell carcinoma occurring in a gland. Think of it—nine years! And it remained in that position all that time.

The next matter that attracted our attention was that in the carcinomata of the upper abdomen, particularly of the stomach, —and you will remember that attention was called to this fact very early by the late Dr. Fenger,—there occurs early an enlargement of the lymphatics just above the clavicle. In order to have an epithelial cell travel from a carcinoma of the stomach to the gland above the clavicle it had to pass through five filter glands before it arrived in that position. How long was it in arriving at the first, second, third, fourth, and fifth filters? From the time that it reached the supraclavicular space up to the time of the patient's death there was very little change in the gland; how long had the metastasis been in that gland before it attained a size that made it recognizable? That again makes us ask the question, How early does transmission take place? If we could answer that question, we would have a more rational or definite basis on which to base an opinion as to when we remove cancers, early or late.

Our clinical experience has taught us that the smaller the carcinoma, the less likelihood is there to be a recurrence. That is both true and untrue. Take, for example, a carcinoma occurring as a small nodule in a rather plethoric woman, at the age of thirty-eight to forty-two, and remove it. Inside of three months you will have evidences or manifestations of recurrence in many places in the body, and before the patient's death you will have numerous metastases, but not a return *in loco.* So disheartening is that particular type of case, occurring in a rather stout woman with carcinoma of her breast at forty-three, that I believe these patients will live longer and live better without the removal of the breast.

Where do they recur? To show you how little we know about it and how few direct points of evidence we have, they will recur in the femur, in the spine, and in the brain. I had in this hospital at one time four cases of bone carcinoma secondary to carcinoma of the breast. Two of these patients came to me primarily because of the bone carcinoma. They came because they had trouble in their bones. One of the patients was a girl who, while playing tennis, strained herself in reaching for the ball. The pain in her thigh became very severe, and finally she came because she thought she had a sciatica. In going over her in the regular systematic way we found that she had two small nodules in her breast, one on each side, not so large as an English walnut. The skiagram showed that she had a softened zone in the upper end of the femur, an inch and a half below the tip of the trochanter. It went on to a pathologic fracture, and she went on to a cancer death without the removal of either of her breasts. There was no occasion for the removal of her breasts when she had a metastatic carcinoma in the femur.

One of the other patients came because of pain in the hip-joint which had been called rheumatism. In looking her over we found that she had an aplasia of the left breast. She insisted that the breast had not changed since she was a young woman. The breast was very small and hard. She had never had pain in that breast, and she did not believe that there was anything wrong with that breast. A skiagram showed that she had a carcinoma in the femur just at the base of the trochanter.

A third case was that of a young woman who came in because of a small nodule in her breast. She was forty years of age, robust, healthy, and vigorous. She had an extensive operation performed and in three months she had pain in her spine, low down; then in her femur, and finally had a metastasis in her brain. She died of metastatic carcinoma some nine or eleven months after we first saw her.

The fourth patient was the mother of one of the most distinguished medical men of this city; she came to us with carcinoma of moderate size in one breast. Her son had recognized the condition practically early. The woman had complained of pain in her back only two or three days before we saw her. While in the hospital we prescribed for her "lumbago." She went on, her spine bent and bent, and she became paralyzed. Another deposit occurred in her femur, and she finally had a pathologic fracture and died from metastases. You can see with that number of cases in the hospital at one time how appalling it is to one when he thinks of these distinct metastases that occur while the lesion is yet comparatively small at the primary location. But you have as a contrast to that another picture—that of the patient who has a small, firm, scirrhous, hard neoplasm, the tissues being coffer-dammed by connective tissue. That coffer-damming connective tissue has contracted and hardened around every erratic epithelial cell. These patients go on for five, six, seven, eight, or even ten years without inconvenience. You remove the tumor by a minor operation on the breast, and the patient remains permanently cured. That shows the greater resistance offered by one class of patients, not a different disease at all in our opinion, but a different resistance to the disease by the individual, and a lesser, weaker, atrophic, contracting, or diminishing, or whatever you care to call it, lymph-supply that comes with age. We know lymphangitis occurs much less frequently as we advance in years than it does when we are younger, with exactly the same cause operating. Of course, we have that demonstration of lesser resistance occurring with other types of infection in which we rarely ever see carcinoma. For instance, in the arteriosclerotic patients there is a different resistance to infections from that found in the average patient in the average condition. But the same question can be put—How soon after the penetration of the basement-membrane are the cells carried either into the blood-current or to the nearest lymph-node, or through the lymph-sieves into the circulation, and then disseminated throughout the body? Until we can answer that question it seems to me we are not going to make great progress as to the definite reason for recognizing carcinoma "early" or "late."

INTESTINAL OBSTRUCTION (SARCOMA OF KIDNEY?).—CARCINOMA OF THE SPLENIC FLEXURE OF THE COLON

HERE is a little child, about five years of age, which we were called to see the other night for intestinal obstruction. The patient did not have a bowel movement for five days. The child had eaten its dinner and went out to play. She came in from play screaming with pain and had nausea and vomiting. The child is pale. It is a typical and classic picture of an intestinal intussusception. The patient was brought in here on the fifth day of the trouble. No bowel movement for five days, and it had vomited only twice, the doctor says. That settled the diagnosis so far as intestinal obstruction was concerned.

In every case of mechanical obstruction of the intestine there is emesis, increasing in frequency, and when the 'phone message came I said, "Has the child vomited?" The doctor said "No, the child has not vomited for three days." It was certain at once that that was not an intestinal obstruction—it was not an intussusception: it was some other lesion. We examined the child, and the abdomen was very much more distended than you see it now, and did not show the beautiful contrast picture. You never see an intestinal obstruction in which half the abdomen is perfectly free except in rotation of the large intestine, occurring in children and adults, with the condition of megacolon. In the condition of megacolon you often see a very large mass in the abdomen. I had one case in which the megacolon contained 56 pounds of feces.

In looking this child over and examining it that night we could get well under an edge, and an edge is always an important thing in the abdomen—a sharp edge. We asked the father if the child had been perfectly well up to this time. At first he said "yes," but finally he said that on two or three different occasions the child had passed blood in the urine. *That told the story.* It was an acute obstruction—an acute retention with either a cystic or a sarcomatous kidney. We will wait until the acute symptoms subside before we make an exploration.

It seems more like a case of sarcoma of the kidney. There was blood in the urine on two or three different occasions, and hematuria rarely ever occurs in a congenital cystic or acquired cystic kidney in this age. In the case of sarcoma and hypernephroma blood is usually found in the urine. The child is too young to have a carcinoma. There is another type of neoplasm—the endothelioma—which may occur at this age, and which may resemble a sarcoma in its clinical course, but it is a true endothelioma.

The next question is, if this is a case of sarcoma of the kidney, what will you do? Would you say to remove a tumor of that size from a child five years old? The tumor goes clear down into the pelvis, and its removal would involve considerable hazard. No! It is wonderful how well these children stand the removal of these neoplasms, but there is not recorded a single case of permanent cure of sarcoma of the kidney in a child where the diagnosis is unquestioned. In every case, as I told you a few days ago in speaking of sarcoma of the testicle and ovary, there is a recurrence of the sarcoma in the other kidney. Just as soon as you take out the affected kidney a growth rapidly forms in the healthy kidney; whereas if the affected kidney is not removed, the other one may remain quiescent for years or until you disturb it.

I recall a patient who was sent to me years ago by Dr. Earle. I examined him, made a diagnosis of sarcoma of the kidney, and advised against operation. The patient went along for a year and had one or two hemorrhages, but there was no material change in his condition. He went along another year and another year, and finally, at the end of five years, he returned to me. I felt then that this could not be a malignant neoplasm, and that, therefore, it should be removed. I thought that it was probably of the hypernephroma type and could be removed.

We removed the kidney without any particular shock to the patient, though it was larger than the one in this child. On examination it was found to be an endothelioma, and in ninety days the patient was dead with a similar growth in the other kidney. That gives you an idea of the course of these cases. This man went along for five years with his tumor, feeling fairly well, but when we removed his affected kidney the growth appeared quickly in the other kidney, grew like a mushroom, and killed the patient in ninety days. The moment you throw additional labor on the other kidney it starts up the process and another endothelioma grows in the other kidney and terminates fatally, with alarming rapidity. The patient was never really well from the day he was operated on until he died.

Therefore in the case of this child the only operation that will be done will be just of an exploratory nature. If it is a cystic kidney, then we must carefully examine the kidney on the other side and ascertain whether it is normal, because congenital cystic kidneys are often bilateral, and if they are, one should be very slow to remove the kidney that first attracts attention.

Now, doctor, please read the history of the patient we will operate on now.

HISTORY

Mr. F., aged forty-two, mail-carrier, admitted to Mercy Hospital May 7, 1912, because of pain preceding and during bowel movements. Family history negative. Had gonorrhea at twenty-seven, with orchitis and epididymitis as a complication, also a urethral stricture, for which sounds were passed repeatedly. Denies syphilis. Had typhoid at twenty-one, with good recovery. Was operated on eighteen years ago for inguinal hernia and undescended testicle on right side. The testicle was removed—on account of atrophy, the patient states. Seven years ago an operation was done on his left kidney for stone. Blood in urine was the only symptom preceding this operation. Skiagram showed a shadow of stone in the kidney, but there was no stone found at the operation (done by a prominent surgeon).

DR. MURPHY: That was an undescended testicle, was it not?

THE INTERN: Yes, doctor.

DR. MURPHY: And should always be removed, because so many of these testicles become sarcomatous.

Dr. Murphy: An additional skiagram was taken, which showed, what?

The Intern: A shadow.

Dr. Murphy: Yes, that is an important point.

Five years ago a second operation was done on this kidney. For the past three months the patient has had colicky pain in the left side of the abdomen, of the spasmodic type, and always followed by a bowel movement. Hemorrhages into the bladder stopped after the second operation.

Dr. Murphy: What effect did the removal of the stone have?

The Intern: No effect.

Dr. Murphy: Oh, yes it did. My dear doctor, it had the effect of stopping the hemorrhage from his bladder. That is a pretty good proof that the doctor did take out a stone; it relieved him of the hemorrhage, and he has not had any hemorrhage since. But he had pain in his left hypochondrium. Put that in, doctor, because it fills out the cycle as it should be, and throws light on his present trouble, and why we are making this exploratory laparotomy.

COMMENTS AND OPERATION

The history shows that he was relieved of his renal complication at the time of operation, and that he now has a new train of symptoms. First, he has pain in the left hypochondrium; following that pain he has a bowel movement; to use his own expression, he has "a bowel movement with pain." When you analyze that you find that he first has the pain and then the bowel movement. He says, "When I have pain I can always feel a mass in my left hypochondrium."

When we examined him yesterday we kneaded his abdomen and suddenly there appeared a mass. Whether it is a fecal retention or a flexion or fixation of the bowel, from the fact that his kidney has been operated on twice, or whether it is a lesion in the lumen of the bowel, I do not know. But I do know that the cycle of symptoms is the cycle of intestinal obstruction of the obturator type. It is periodic, recurrent, and increasing in frequency. These are the salient things about that history:

First, all his renal symptoms are wiped out by the last operation; second, the urine examination is negative, showing that he has no renal trouble now; third, he has these pains in a definite place—they are of the spasmodic type, and associated with his bowel movements; fourth, when this pain attains its greatest degree of severity he hears a gurgling. That is the return gurgling that comes after a spasm of the bowel, and it is characteristic of that condition.

Now, we will endeavor to find the cause of his trouble on the left side. I could not feel the tumor except after excitation. What it was that produced it we will try and show you today.

I am going to open the abdomen by making the incision at the outer margin of the left rectus muscle, near the linea semilunaris. I will cut through the skin and superficial and deep fascia until the anterior aponeurosis of the left rectus muscle is exposed. This aponeurosis is split so that we can displace the rectus muscle inward and pass through the posterior layer of the rectus sheath into the abdominal cavity. By this procedure the opening in the aponeurosis and that in the peritoneum will not be on the same line. Next, we incise the peritoneum and expose the intestine. We will examine the intestine carefully, searching for the mass which we palpated through the abdominal wall. Here it is in the transverse colon. It is a carcinoma—a carcinoma of the large intestine. Our analysis of the symptoms was correct. We could not feel this tumor until the spasm of the intestinal muscle brought it into prominence. We will do a resection of the bowel and make a lateral anastomosis. The first step is to expose the zone of the tumor in the mesentery. Now we will commence to ligate the mesocolon and expose the zone of the tumor. I am ligating this mesentery, and with a few ligatures above so as to free the upper half and with a few ligatures below so as to free the lower half, from its attachment. We free it for a number of inches both above and below. We will examine the mesentery carefully for enlarged lymph-nodes. There is just one node involved. It contains a large metastatic carcinoma. We palpate the mesentery a little more to see if there are any more nodes involved. There seems to have been only that one enlarged gland in the entire zone. Now I am in the dead space, through which I will pass my ligature through the mesentery. I will pass along over that zone and get beyond the line of metastases to the next dead space, where I will put on the next ligature. That will allow me to remove about six inches of the intestine. I have to go as far away as that so as to keep away from the vessels.

In operating on the large mesentery always use the angiotribe or crushing forceps. That makes a crease into which the ligature fits, forces out the fat, and when I cut off the mesentery there remains a ledge on the end over which the ligature will not slip. I have in the bite of the forceps practically only blood-vessels and the peritoneum of the upper and lower margins.

I will ligate this mesentery on the distal side, because otherwise I would have a bleeding from that. This is only a temporary ligature, because all of that zone is to be excised.

This patient has had repeated attacks of obstruction, and that has caused the overdevelopment of the muscle layers of the bowel, which caused that tumor which we recognized yesterday, and which appeared after we irritated the bowel and produced contraction of the muscularis. We are now ready to divide the intestine, and the method by which we divide the large intestine is practically identical with the method by which we divide the appendix.

I lift the intestine up into the field and find the portion which I am going to resect. I will put that intestine into this angiotribe; that forces the mucosa of the bowel out of the bite of the ligature, just as in the case of the appendix. Then I divide the bowel with a knife, cauterize and sterilize it, and then invaginate it, just as I would an appendix. With a fine straight needle threaded with silk I pick up the proximal end of the bowel at the mesenteric side first, because that is always the danger-point so far as bleeding, infection, and leakage are concerned, because there is no peritoneum over that zone. I will turn that end into the lumen of the bowel. There will not be bleeding into the bowel. Why? Because I have cauterized the bowel; it will not suppurate at its end because I have sterilized it. This proximal end is the most dangerous end. Why? Because this leaves a blunt end on the proximal portion against which the pressure of the intestinal contents will come. For that reason we will insert here, in addition to the ligature, two rows of continuous linen sutures. That closes the proximal end of the bowel safely and securely.

In the distal portion there is an excess of tissue; there is our neoplasm. I am going to excise it with about six inches of intestine, but I will first make a lateral intestinal approximation. The line of union will be near the splenic flexure. The splenic attachment is high up, in the usual way. We must free these bands a little and let the bowel come down.

I am going to make the division of the bowel close to the splenic flexure, but I will make the approximation before I excise any bowel. After I have made the attachment I will amputate. There is the omentum coming over from the side; we will later fold that around the line of apposition. I make this union by the regular continuous suture. The angiotribe is not put on the distal portion of bowel beyond the line of division. The first suture which I will pass through here is a continuous suture including only the fibrous or serous layer of the intestine. First we put in the posterior Lembert sutures, then we put in the Czerny sutures without opening the bowel until we have put in the return series of stitches. These two sutures go through all the coats of the bowel and they are inserted parallel to the long axis of the bowel. We pull them fairly taut and place the stitches close together, so as to lessen the likelihood of bleeding into the bowel.

I commence to make my first return stitch, just where I am going to divide the bowel. I am using the Moynihan needle for that. I have completed the Czerny portion of the stitch, and I am going back and do the Lembert portion. The two portions of bowel are then incised so as to establish continuity of the lumen. We complete the anastomosis by continuing the sutures around the openings into the bowel in the usual manner.

Now we are ready for the amputation of the distal end. The intestine is surrounded by strips of gauze and the field of operation is walled off, just the same as we did at the other end. The clamp is applied and the intestine is severed so that we can now remove the tumor mass. We place a permanent ligature on the invaginated portion in the groove made by the clamp to avoid hemorrhage. This end will have the least tension put on it because it is distal to the approximation and distal to the bowel that will carry the feces on down past the line of approximation. Having completed the intestinal union and securely closed both ends of the bowel, I will fasten the omentum over the line of union with a few fine catgut sutures, so as to anchor it. That gives additional security against leakage and infection. The late Dr. Senn accentuated the importance of this procedure, and I think he was perfectly justified in doing so.

Having replaced the intestine in the abdominal cavity, the next step is to suture the peritoneum by making an ectropium of the cut edges, turning them out and thus preventing the formation of intra-abdominal adhesions. A continuous catgut suture is used for this purpose.

The incision in the posterior and anterior sheaths of the rectus muscle are closed with catgut. The deep tissues of the abdominal wall are approximated and sutured with a silkworm-gut figure-of-8 stitch; the skin is closed with horsehair. The wound is dusted with bismuth subiodid and covered with a gauze collodion dressing and a large abdominal pad over all.

In all these cases we put in a rectal tube. Why? To prevent the accumulation of gas, so there will be no tension on the intestine.

Let us examine the specimen. It is a carcinoma, not a hypertrophic tuberculosis. There are no caseous deposits. There is a stenosis of the bowel. What was the report on the feces examination, doctor?

THE INTERN: Blood was found every time.

DR. MURPHY: Every feces examination showed blood. Let us connect the pathology with the clinical course of the disease. We were told that the stools were formed in a ribbon-like way. That is a misleading statement, because it leads one to believe that there is some obstruction at the outlet. The intern's version of the case fits an obstruction lower down. When the feces were passed, blood was on the surface, rather than mixed in with the mass. What was said about the pain relative to the bowel movements? The pain was followed by a bowel movement, but the most important thing is that when he had pain he also had the formation of a tumor; he told us the story before we saw the tumor. He had the formation of a tumor in his abdomen on the left side. He said, "When I get a real cramp I can feel the tumor, and as the tumor reaches its greatest size and the pain the maximum degree of severity, I hear the explosion of gas." That is typical; it comes along harder and harder, then finally ceases, and then comes the explosion—you hear a gurgling.

I remember a case which I saw many years ago. We kept the patient under observation for about four weeks, and every day, when we gave her a full meal, the gas came up to the obstruction, and on placing the stethoscope over the tumor one could hear an explosion; the pain would cease, and she would have no more pain until we gave her another full meal. She had these attacks of obstruction in the small intestine for three years. The operation showed an opening or lumen in the bowel a little larger than the lead in a lead-pencil, and an annular stricture of the small intestine just above that point. It was enormously enlarged.

STUDENTS' CLINIC—FRACTURES

[We have received many requests from our subscribers to publish, in one of the issues of the CLINICS, a report of the clinics which Dr. Murphy holds for the senior class of students at the Northwestern University Medical School. In accordance with these requests we publish below a verbatim report of a students' clinic, feeling assured that this will prove interesting to our readers.—PUBLISHERS' NOTE.]

DR. MURPHY: At the last clinic we had under consideration the subject of fractures. What do you understand by a compound fracture?

FIRST STUDENT: A compound fracture is one where the bone fragment penetrates the skin or an opening is made from the skin or mucous membrane to the point of fracture.

DR. MURPHY: And the fractured bone is visible externally or has been retracted inward?

FIRST STUDENT: Yes, sir.

DR. MURPHY: Into what classes do we divide fractures?

FIRST STUDENT: Into simple and compound. A compound fracture communicates externally, directly or indirectly, and a simple fracture does not.

DR. MURPHY: Does that make any special difference?

FIRST STUDENT: It makes a difference in the treatment.

DR. MURPHY: Why?

FIRST STUDENT: Because there is liable to be more infection in a compound fracture than in a simple fracture.

DR. MURPHY: And that infection in a compound fracture is known as what type of infection?

FIRST STUDENT: Direct.

DR. MURPHY: And the infection in a simple fracture is indirect or what?

SECOND STUDENT: Hematogenous.

DR. MURPHY: What do you understand by indirect infection in a fracture?

Second Student: Infection through the blood-stream.

Dr. Murphy: How does it get to the fracture?

Second Student: There is an extravasation of blood into the gap between the fragments of the bone; bacteria that have gained entrance to the blood-stream are lodged at the site of fracture.

Dr. Murphy: What is that?

Second Student: Bacteria gain entrance to the blood-stream and lodge at the point of least resistance or point of internal laceration.

Dr. Murphy: With the extravasation of blood there occurs escape of bacteria from the blood-stream into what?

Second Student: Into the clot at the point of separation of the bone.

Dr. Murphy: How can that effect be produced artificially?

Second Student: By trauma.

Dr. Murphy: What do you mean by trauma?

Second Student: An injury, such as a blow or an operation.

Dr. Murphy: You have not figured that out very well. What have you to say, sir? [Referring to the third student.]

Third Student: When one breaks a dog's leg and a nidus of infection has been created somewhere else in his body, there may be bacteria free in the blood and they will lodge at the site of fracture.

Dr. Murphy: Particularly if you do what?

Third Student: If you inject bacteria before the injury.

Dr. Murphy: Because they are circulating in the blood?

Third Student: Yes, sir.

Dr. Murphy: What filter have they passed through? How do you get infection in a rabbit's leg?

Third Student: The bacteria have already passed from the lymphatics into the blood-stream. They have broken through the lymph-glands.

Dr. Murphy: No, not at all; not at all. If you inject bacteria into the blood-vessel of a rabbit's ear, how does the rabbit get an infection in his leg?

Third Student: The lymphatics gather up the septic material and carry it to the venous side of the circulation and then to the arterial side, and from there to the leg. The septic material in the vein of the rabbit's ear passes first into the right side of the heart, then into the first filter, the lung, and if not arrested, into the left side of the heart and on into the general circulation.

DR. MURPHY: And after it passes through the capillaries of the lungs it can be arrested where?

THIRD STUDENT: It can be arrested in the heart, left side.

DR. MURPHY: In what zone?

THIRD STUDENT: In the arterial zone.

DR. MURPHY: In the general circulation zone, is it not?

THIRD STUDENT: Yes, sir.

DR. MURPHY: That is right. If you inject bacteria into the cellular tissue, they are taken up by the lymphatics before they get into the blood-stream, and after they get into the blood they pass through the lung first. Do we have infections of bone occurring in any other way except accompanying fractures?

THIRD STUDENT: Yes.

DR. MURPHY: In what disease?

THIRD STUDENT: In the disease known as acute infective osteomyelitis, tuberculosis, etc.

DR. MURPHY: What is the difference in the process of repair between a simple fracture and a compound fracture, and why?

FOURTH STUDENT: In a compound fracture you have the clot disturbed as it is formed when there is infection present. In a simple fracture, after the extravasation of blood has occurred, fibroblasts begin to form new tissue, new capillaries are being formed, and healing proceeds with the formation of new bone. In a compound fracture there is infection, which prevents healing. by destroying the embryonal tissues as they are formed.

DR. MURPHY: When there is no infection, what is the difference?

FOURTH STUDENT: There is no difference whatever.

DR. MURPHY: What is that?

FOURTH STUDENT: The only difference, then, is the extent of time.

DR. MURPHY: In the infected fracture no clot is formed. There is a difference, then?

Fourth Student: Yes, sir.

Dr. Murphy: After infection, if a clot forms, there is a dissolution of the clot by the infection, and still, for a time, granulation tissue forms. The clot breaks down entirely, does it not, and escapes as an exudate?

Fourth Student: Yes, sir.

Dr. Murphy: In time it is walled off?

Fourth Student: Yes, from the end of the bone.

Dr. Murphy: How?

Fourth Student: By granulation tissue forming.

Dr. Murphy: Where?

Fourth Student: At the end of the fragment.

Dr. Murphy: In every injured tissue, in every tissue that is lacerated, there immediately commences what process?

Fourth Student: The process of repair.

Dr. Murphy: Close to the line of injury or to the line of trauma the tissue becomes necrotized, and the effort at regeneration commences at the line of demarcation between the living and the dead tissue to separate—what?

Fourth Student: The living from the dead.

Dr. Murphy: Go on, sir.

Fourth Student: In a simple fracture, after a clot has formed, the capillaries from the Haversian system and the medullary canal and the periosteum bud out to form granulation tissue out into the clot.

Dr. Murphy: And the clots acts as what?

Fourth Student: As a scaffolding.

Dr. Murphy: Correct.

Fourth Student: The fibroblasts come in from the periosteum, forming fibrous tissue. Then, about the third week, the osteoblasts come in from the osteogenetic layer of the periosteum.

Dr. Murphy: Of what?

Fourth Student: From the osteogenetic layer of the periosteum.

Dr. Murphy: The clot is first displaced by what?

Fourth Student: By granulation tissue.

Dr. Murphy: And subsequently by fibrous tissue—by newly formed connective tissue. That is what you mean by fibrous tissue. That constitutes what is known as the temporary soft callus. Union of the ends of the bone is effected by the soft callus. And there comes next—what?

FOURTH STUDENT: The process of ossification.

DR. MURPHY: Yes. Where are the osteogenetic factors situated?

FOURTH STUDENT: In the osteogenetic layer of the periosteum and in the medulla.

DR. MURPHY: Let me give you an example, so that you can never forget where the osteogenetic forces are located. In speaking of the osteogenetic forces and where they are situated they may be likened to the trunk, branches, and leaves of trees. The trunk of a tree represents the medulla. It is the most potent osteogenetic force or tissue in the body at *all periods* of life. Next to that come the Haversian canals, which have elongations of the vessels both from the periosteum and from the medulla. Next come the lacunæ and then the canaliculi, and at the ends of the canaliculi you have spread out the leaves of the osteogenetic tree, represented by the deep osteogenetic layer of the periosteum, which in the springtime of life is enormously potent in the production of bone, but late in life has little potency in the reproduction of bone. Therefore the osteogenetic forces are situated, first, in the medulla; second, in the extensions of the medullary cavity, which are the Haversian canals; third, in the lacunæ, which are extensions of the Haversian canals; fourth, in the canaliculi, which are the extensions again of the lacunæ, and fifth, when they come to the surface they spread out under the periosteum,—the deep layer of periosteum,—representing leaves. You cannot forget that. These positions which I have mentioned represent the locations of the blood-vessels from the center to the periphery of the bone. These blood-vessels carry with them always two cell-controlling elements—the osteoblasts and the osteoclasts. The osteoblasts are the builders of bone, while the osteoclasts remove the dead and unnecessary bone.

Now, when a bone is fractured, regeneration occurs from all five of these positions, varying in degrees at different ages: most potent, at all times, in the medulla; next in frequency and of greatest potency, in youth, in the periosteum; then in the Haversian canals, then in the lacunæ, and then in the canaliculi. That is a simple matter. You surely can never forget it. On the wall of the blood-vessels are osteogenetic cells known as the osteoblasts, and there are also found the osteolytic cells known as the osteoclasts. Therefore, when a soft callus is formed there commences immediately the reproduction of bone proceeding from all five sources I have mentioned, primarily and most readily, as in the growth of the leaf of a tree, from the periphery or the periosteum. Next, in the deposits in the medulla, and then in the intermediate spaces between the medulla and periosteum.

You have, therefore, callus divided into three distinct sets: First, and most early formed, the subperiosteal callus; second, the medullary callus; and third, the intermediate or definitive callus, the one that is to remain. In normal adults, three and a half weeks after the fracture occurs, a bony callus begins to form in the subperiosteal zone; in five weeks it is formed in the medullary area, and in about six and a half to eight weeks it forms in the intermediate zone between the compact bony ends, and that goes on to complete ossification and to the production of a bony callus sufficiently large to give ample support to the weight-bearing force required in that particular bone.

When that is complete, there commences another process—the process of absorption of the excess of callus, and that process of absorption proceeds in exactly the same order as does the process of production of bone. The absorption commences in the periphery, and all the excessive callus is absorbed except where it contacts with other bone tissue and ossification has taken place. Next, the excessive callus in the medulla is absorbed, and there is effected a complete reëstablishment of the medullary canal, and there remains permanently the definitive callus between the compact bony ends.

That is not difficult to understand. It is essential for you to know these elementary principles if you are ever going to understand the process of repair of bone following fractures, if you are ever going to understand the process of regeneration of bone—

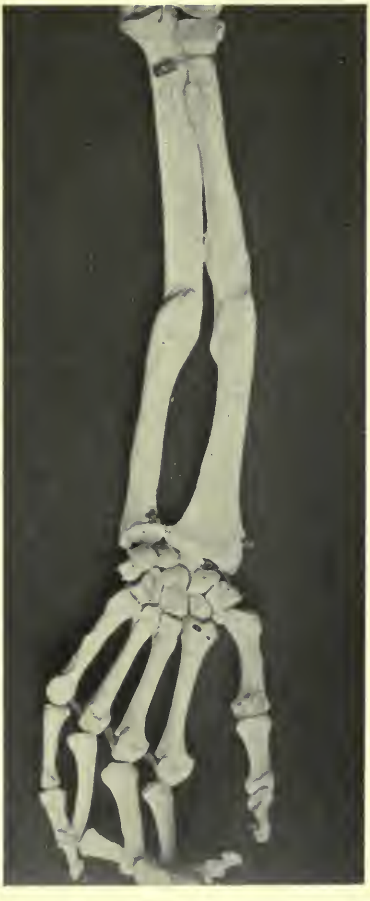

Fig. 118.—Photograph showing fracture of ulna and radius, with good union. Little deformity and good functional results. One view.

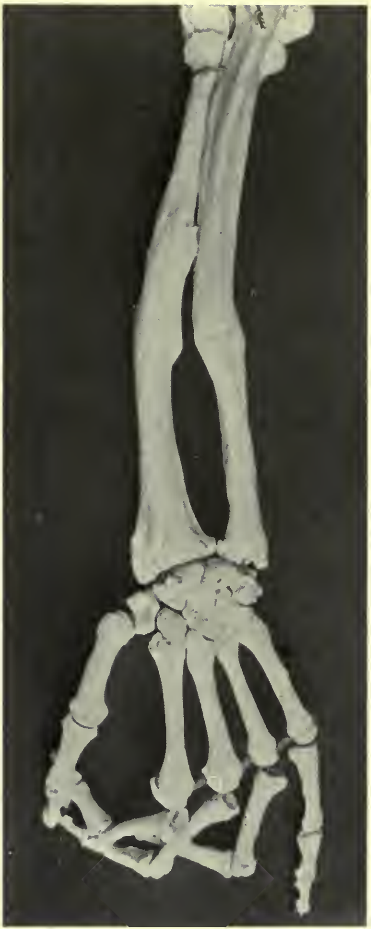

Fig. 119.—Photograph showing fracture of ulna and radius with good union. Little deformity and good functional results. Another view.

if you are ever going to analyze and explain to yourselves why in certain cases of fracture there occurs union and why in others there is failure of union. It is very essential for you to know these things if you are going to understand why you resort to certain operative procedures to effect union after primary union of the bony structures has failed. All these things must be clearly settled in your minds. In medicine the first question is—why? You cannot forget if you know why. It is your property when you know why. If you do it in a parrot-like way, without knowing why, it is the other man's property and not your knowledge at all.

In the repair of fractures we often meet with beautiful illustrations of the results following a simple fracture, on the one hand, and a compound fracture, on the other. Just look at these two pictures. Here (Figs. 118 and 119) is a simple fracture that has healed with some deformity. There is some deviation in the contour of the bone—just a little, but the patient has a serviceable limb. Healing has taken place by primary bone repair. Here (Figs. 120, 121, and 122) is a picture showing a compound fracture with considerable deviation of the plumb-line. If you analyze this specimen with a plumb-line drawn through the trochanter to the intercondyloid notch in front or behind, you will see that, notwithstanding the enormous deviation shown there, this man had, when he walked, practically a perfectly straight and serviceable limb. Think of it! You see why. Run a line down on the side and see where it strikes. The bone at the union of the fracture falls in front of the line. There is a marked angular deformity. And yet he had a serviceable limb even with the colossal displacement at the bone ends.

Here we have an illustration of the rôle which a separated fragment of bone plays in bridging over the gap or distance between the fragments in a compound fracture. You can see in this case that this fragment was separated from the proximal and distal bone-ends, but it never lost its vitality; it retained its periosteum and lived. Fragments of this periosteum were carried around here in various positions, and that supplied new osteogenetic elements which finally filled in the defect and gave the man sufficient support for his limb, acting as a trestle-work or resembling the cantilever plan. It shows how potent these osteogenetic elements are, even under unfavorable conditions, namely, those of infection. It must have taken a long period of time to produce that result, as shown by the excessive callus deposited here.

In other fractures, even where the fragments are placed practically in accurate apposition, there is not the slightest effort at the reproduction of a callus. Look at this picture! Note the splendid position obtained by that Lane bone-plate, with perfect apposition, but no effort whatever on the part of—what? On the part of the osteoblasts to reproduce the bone. Contrast these two pictures. See the enormous osteogenetic force exercised in the one, and the absence of the osteogenetic force in the other. In this case it would do no good whatever to put in another plate: you could not get better apposition than is already procured. What you want to do is to stimulate the reproduction of bone; it can be most effectually accomplished by transplanting a fragment of bone from the patient's tibia and inserting it into the medulla, spanning the gap between the separated ends—some 4½ inches in length. Always use an auto-bone graft, as homo- and hetero-transplants are practically always failures.

That bone merely acts as does a blood-clot. For what? For a scaffolding over which the Haversian vessels may pass from above and from below, passing through these canals, and carrying with them the osteoblasts and osteoclasts, which, in time, will remove all that transplanted fragment, but when they do, the fragment will be replaced by another bony framework of exactly the same mold and size. Finally, that new bone will increase in size in its periphery to support any strain that is placed on it. It is really a physiologic hypertrophy. This is one of the natural results of stress placed on any weak point in the body. You see why we put in that bone transplant. You see why you have repair here, and why there is no effort at repair, even when the bone-ends are brought in apposition, without contact of freshened bony surfaces so that the osteogenetic force can be brought into play and do their part of the work.

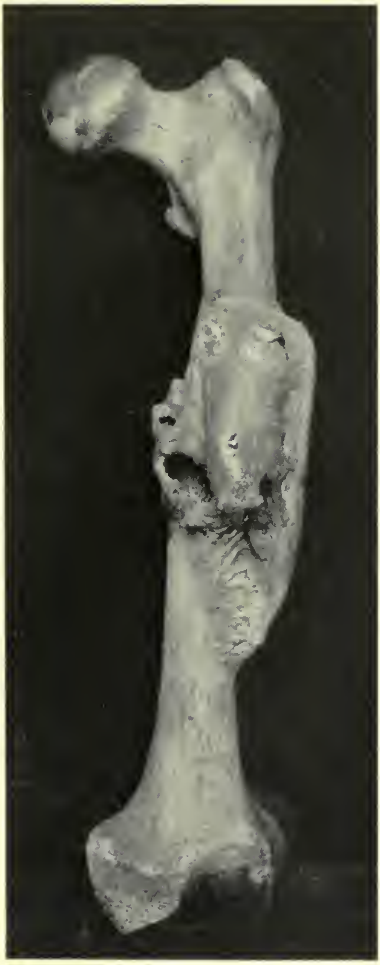

Fig. 120.—Fracture of left femur (anterior view), showing a fracture with union, but formation of large amount of bony material. Apparently no deformity.

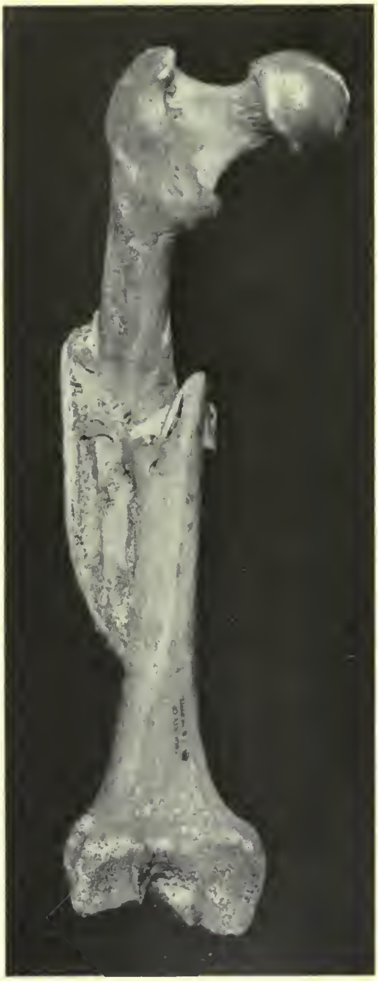

Fig. 121.—Same case (posterior view), with lower fragment pointed and directed slightly away from end of and inward to upper fragment.

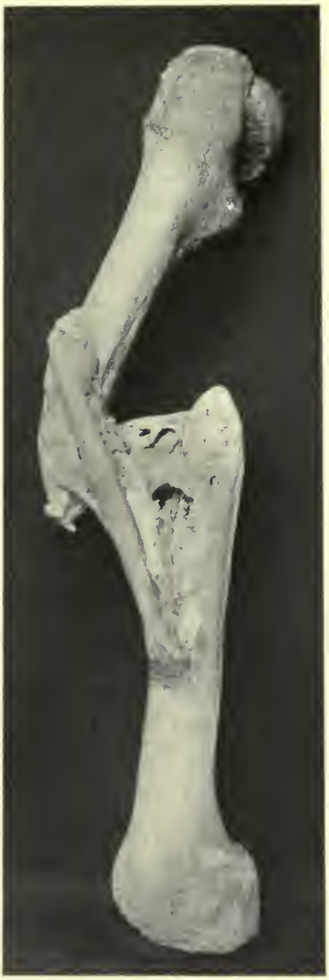

Fig. 122.—Photograph showing marked deformity. Same case (lateral view) showing marked deformity, with maintenance of line of gravity so that patient had normal function of leg so far as usefulness is concerned. This deformity was not apparent in the other photographs.

In a compound fracture there follow the outpouring of blood, the formation of a blood-clot, and then the process of repair can go on just the same as in a simple fracture if infection does not take place. Therefore in every compound fracture the first and most important thing is—what? To protect it from sepsis; to protect it from infection. The way in which it is protected from infection is first to neutralize or sterilize the field around the wound with iodin immediately after the injury; second, to place the fragments in the best position you can without opening the wound, if possible.

If you do open the wound to replace the fragments in apposition with the aid of forceps, avoid all additional trauma at the time of the operation, and, above all things, never endeavor to put in a foreign body to maintain immobilization or to secure fixation immediately after the occurrence of a compound fracture. If you do, according to my personal observation, extending over a period of about thirty years, the result, in an enormous percentage of the cases, will be that they will become infected, and eventually you will have to take out the foreign body. It becomes a detriment in place of an advantage.

That is one of the most difficult things for us to learn or to unlearn regarding a compound fracture. A great deal depends on the way you put in the foreign body. That additional handling will enable a sufficient number of the saprophytes and pyogenic bacteria to produce infection locally, and therefore the foreign body is a detriment. On the other hand, if you let the tissues come together with the least trauma, approximate the edges of the wound with suture, if you can, immobilize the leg, and wait until nature heals the soft parts, the chances are that you will get a good result. If, in the case of a fracture of the femur, the fragment should overlap an inch or two, it does not matter much, for you can put on Buck's extension and overcome the shortening. If it is in the lower leg, you can put on a Buck's extension and overcome the shortening. But immobilize the tissues first, and if you keep everything out of the wound,—fingers, instruments, and other foreign bodies,—you will, in all probability, get primary union.

At the end of three weeks, when primary union is complete, if there is still overlapping of the fragments of two or three inches, then, with the field prepared, expose the ends of the bone, put on traction force, after the plan recommended by Dr. Edward Martin, of Philadelphia, elongate the limb to the necessary extent, and maintain approximation, if necessary, with a Lane bone-plate and intramedullary bone splint from his tibia after Murphy, or in any other way you may choose, close the wound, and primary union will take place, the process of bone repair scarcely having been retarded a day. Why? Because the soft callus that has formed during the three weeks you have been waiting is ready to go on to consolidation. The case will terminate favorably without the patient having been exposed to the hazard of an infection which is so common with immediate operative procedures in compound fractures.

It is the most difficult thing—and it has been a difficult thing for a quarter of a century—to keep instruments and fingers out of a compound fracture. In my early experience it was the habit of the average practitioner who saw a case of fracture on the street to run to a drug-store, wash his hands, and stick his fingers in the wound to see how badly the bone was fractured. What followed? Infection!

When I was a surgeon at the Alexian Brothers Hospital the first question asked by the intern was, Has this wound been examined? That is, was it examined before the police brought the patient to the hospital? If not, we could figure that 80 per cent. of the cases would go on to primary union without infection. If the patient had been examined, you could figure that the patient would have 80 per cent. of chances of infection, because the microörganisms were carried into a field whose resistance had been lowered or destroyed by the trauma. The result was that infection followed. Lane's and our experience is that compound fractures should never be primarily plated or wired or nailed or operated.

Case 2.—Before examining this patient I want the intern to read the history of a patient on whom we operated about an hour ago, because the history tells the story. I want you to listen carefully to this history, because it is the story of two lesions occurring in the same patient, one terminating in a perfectly good, useful joint; the other terminating in a destroyed joint, which I removed this morning.

TUBERCULOSIS OF RIGHT KNEE-JOINT

HISTORY

Mrs. C., thirty-five years of age, married. Entered the hospital because of pain and soreness in the right knee-joint.

Family history negative as to tuberculosis. Has always been perfectly healthy until present trouble.

Menstrual history negative. Was pregnant but once; one child, now three years old; well.

Present Trouble.—In the spring of 1902 she began to experience slight pains in the right knee-joint. She thought that they were neuralgic, and paid no attention to them. Knee was not sore or tender, not swollen, and did not interfere with use of limb in any way. Pains gradually increased in severity, and continued, with intermissions, until five years ago. For days at a time she would be free from pain.

About five years ago pain became quite severe, and the joint became swollen and tender. Not until this time had symptoms increased to such an extent that she thought it necessary to consult a doctor. The knee gradually became very tender, and she was constantly on guard lest she strike it against chairs or other objects. It was not reddened, and was not too sore to prevent it being used in walking about carefully, but after using it a great deal it would be stiffer than usual the next morning. For the past year it has been much worse, and she has been able to get about only by the use of crutches. She can feel a crunching, slipping sensation in the joint on motion. Knee is not ankylosed and never has been. It can be bent, only very slowly and carefully, however, without causing great pain. Joint never has been opened for injection or drainage. Knee never has been injured or had any trauma. Muscles attached to knee are not atrophied. No sinus or discharge of pus has ever been present.

Three years after the onset of the trouble in the knee she had a sudden, severe pain in the right ankle, and the joint rapidly became swollen, tender, and reddened. She was sick in bed with a high fever (?) for ten days, but had no chills. She does not remember anything in regard to any exposure or lowering of resistance previous to this, but knows that the attack occurred during cold weather. The ankle-joint got well rapidly, with no ankylosis or even limitation of motion. This attack did not influence the symptoms or course of the disease in her knee in any way. Urine normal. Heart negative.

There are two lesions, you see—one an acute lesion, coming on with a high temperature, swelling of the joint, immediate incapacity, progressing to the stage of inflammation for ten or twelve days, then subsiding and entirely disappearing; the other lesion causing swelling, a little at a time, growing a little worse and a little worse, continuing for a period of nearly five years before the patient was materially inconvenienced by the disease in that knee. It was always painful the day after exercise. Finally, there was an acute exacerbation; the process remained stationary for a while, and then there was another acute exacerbation, again remaining stationary. But always what? Always increasing in severity until finally she comes here with cringing in the knee, great pain, walking on crutches, slight motion in the joint.

The diagnosis in the first case was—what? Rheumatism! What is "rheumatism"? What do you mean by "rheumatism"?

STUDENT: An acute inflammation due to some unknown microörganism.

DR. MURPHY: Tell us about it. "Rheumatism" does not mean very much. It is a nice way of expressing what you do not know.

STUDENT: It begins with a slight fever, swelling of the joints, usually one at a time.

DR. MURPHY: Sometimes.

STUDENT: It usually starts in the knee, then goes into the hip, and down to the ankle.

DR. MURPHY: Sometimes.

STUDENT: It may be confined to one joint.

Dr. Murphy: That is correct—sometimes.

Student: There are usually swelling of the joint, redness of the joint, and tenderness. The patient is usually incapacitated.

Dr. Murphy: What is the matter with the patient?

Student: She is sick.

Dr. Murphy: That does not tell the story, sir. What is the matter with the patient? What is really the matter with this patient?

Student: She has an infection.

Dr. Murphy: What do you mean by that?

Student: That some microörganism in the body is causing infection.

Dr. Murphy: But what do you mean by that?

Student: There is inflammation of the tissues.

Dr. Murphy: So is appendicitis an inflammation of the tissues. What is the matter with the ankle? You said the knee was involved in rheumatism. Let us see where we arrive following the average course, and why we conclude erroneously. Tell us about it.

Student: According to the history, there was an arthritis; then later involvement of the bones of the joint.

Dr. Murphy: What do you mean by that? It is not clear. The other gentleman admitted it was an arthritis. It was in the joint. If he did not use that expression, he intended to do so. But you have not added anything material to the case.

Student: There is a history of slow onset, pain, gradually increasing in severity, and later followed by swelling of the knee-joint, with redness.

Dr. Murphy: There was no redness of the joint at any time.

Student: The course leads to tuberculous involvement.

Dr. Murphy: Do you mean tuberculous "rheumatism"? Do you amend the other student's diagnosis? We do not think it was "rheumatism."

Student: The history points to tubercle bacilli lodging in the epiphyseal line, probably in the joint end of the femur.

Dr. Murphy: Have some kind of precise picture of the pathology in that joint. Have some mental picture of it. If it is rheumatism, where is the lesion? If it is tuberculous, where is the lesion? If it is neither, where is the lesion, and why?

STUDENT: Tuberculous disease locates itself on the joint surfaces of the epiphyseal line.

DR. MURPHY: No, sir, it never does. There is no such place.

STUDENT: The distal portion of the epiphyseal line.

DR. MURPHY: What do you mean by distal portion of the epiphyseal line? I suppose you mean the line of demarcation where the osteogenetic elements are at work in elongating the bone in infancy and childhood up to adolescence. It is the line of division between the circulation of the epiphysis and the circulation of the diaphysis or shaft in the early part of life. It is a trocha, in other words, and of cartilaginous consistence. It is covered above and below with osteoblasts in great number. Now, then, he says this begins on the epiphysis,—the articular surface of the epiphyseal line,—and the epiphyseal line is never on the articular surface. Go on, doctor.

STUDENT: In childhood the tuberculous focus lodges on the shaft outside of the epiphysis.

DR. MURPHY: Yes, on the shaft side? In infancy the epiphysis is cartilaginous. Metastatic infection of any kind never lodges in a cartilage. That is a law.

STUDENT: The tubercle bacillus lodges where there is an abundant blood-supply.

DR. MURPHY: Yes, it lodges where there is a rich blood-supply, and in infancy and early childhood there are many capillary loops on the shaft side of the epiphysis.

STUDENT: The tubercle bacillus lodges and multiplies and gives off toxins which lead to the formation of a tubercle.

DR. MURPHY: The tissue cells multiply and form a tubercle of increasing size. Finally, there occurs an ischemia of the central portion; this dies, becomes necrotic, and a cheesy nucleus is formed. All right, sir, go on.

STUDENT: The tubercle becomes caseous, or fibrous, or calcified. It may break down. Other tubercles may develop. All these may coalesce and form an abscess, which travels in the tissues in the direction of least resistance. In the mean time there are irritation and swelling, due to the irritation of the synovial membrane and infiltration of the connective tissue.

Dr. Murphy: The swelling is due to what?

Student: Effusion.

Dr. Murphy: As the process nears the surface of the joint cavity it causes effusion into the joint long before the tubercle bacilli arrive in the joint. It produces irritation and congestion, and the joint is often congested weeks and months before tubercle bacilli enter or before the tuberculous focus ruptures into the joint. The patient is incapacitated, usually manifesting symptoms of a severity in proportion to the degree of disturbance caused on the granulating, inflamed focus and by the pressure on the joint surfaces when the patient walks or exercises. Go on, sir.

Student: The abscess travels around the joint.

Dr. Murphy: It does not travel around the joint. If the focus continues to enlarge, it ruptures inside the capsule and into the joint cavity. When doing arthrectomies we find that in the enormous percentage of cases of knee disease the tuberculosis begins as a primary bone lesion, and not as a synovial lesion. It is a tuberculous osteitis, and the joint surfaces become involved later after the rupture of the bone focus into the joint. That is our guide and aid in the treatment.

What good will it do, if you have a tuberculous focus here in the femur or there in the tibia, to treat that kind of a joint by injecting any kind of fluid into the joint? None whatever, as the lesion is then in the bone. If the lesion in the knee is a primary synovial tuberculosis, as it sometimes is in adults and many times is in children, and as it is more commonly in the hip and in the elbow, then you can benefit the patient by the injection into the joint of a fluid that increases the number of polynuclear leukocytes, and that stimulates the production of connective tissue around the infected focus. But in the knee you get practically no benefit at all from such injections because tuberculosis of the knee in adults is so commonly a primary bone lesion. You should know why you are doing certain things and why you are not doing others. What was the matter with the girl's knee? Why did you conclude it was tuberculosis?

Student: The rheumatism might be a general septic involvement.

Dr. Murphy: What do you mean? What are you talking about? I am not talking about rheumatism; I am talking about the lesion in the knee.

Student: It first started in the bone.

Dr. Murphy: What symptoms did she have in the beginning? Pain on excessive exercise; a little stiffening, more so when the limb had been exercised severely; no swelling or effusion into the joint in the early stage at all, and at the end of five years she developed a partial incapacity. As soon as she began to use the limb and got it accustomed to walking she had no pain at all. But when simply standing, with her leg in certain positions which produced continuous pressure on the bones of the knee-joint, she had pain. That is not rheumatism. What was it?

Student: Tuberculosis.

Dr. Murphy: Where was the tuberculosis? Either in the femur or the tibia. In this particular case the disease began in the internal tuberosity of the tibia. We found there a caseous mass. It finally ruptured into the joint or extended to the synovial membrane, and then got under the margin of the synovial membrane, burrowed in under, and removed all the joint cartilage. When she entered the hospital she had no cartilage at all in the joint. The two raw bony surfaces were being rubbed together, and it is this which gave her such great pain. There was little, if any, effort at fibrosis; little, if any, effort at resistance on the part of her tissues against the destructive process. She was in a lamentable condition. Often you will find in a joint in which tuberculosis has been present for a period of time an enormous quantity of connective tissue, no definite erosion at all, but a mass of fibrous tissue, with great fixation and limitation of motion in the joint. There is no limitation of motion in this case, because there is no tendency to fibrosis, which is the reparative effort made by the tissues against tuberculous invasion. What was the second lesion—the lesion in the ankle? This case would not give a good result with injections—tuberculin, immobilization, or any other expectant treatment—as the tissues are making no spontaneous effort to overcome the tuberculosis by fibrous tissue production.

Student: It might be the same thing.

Dr. Murphy: Tell me why it might have been so, and why it might not.

Student: The same process is going on there.

Dr. Murphy: Tell me about it. Was it the same process as in the knee-joint?

Student: I think so.

Dr. Murphy: How erratic and inconsistent that kind of a statement is. One was a lesion that took five years to incapacitate the knee; the other was a lesion that took five hours to incapacitate the ankle, and still you say it was the same infective process.

Student: It is only three years ago since the ankle became involved.

Dr. Murphy: Justify your position. The patient was immediately incapacitated. Defend your position. I think you are in error. I know you are in error.

Student: I did not get the point about five years and three years ago.

Dr. Murphy: The patient was immediately incapacitated, as I have said. The patient had a temperature of 103° F., swelling of the ankle-joint, was intensely sick for ten days and then the process subsided entirely and the ankle-joint was just as good as it was before. That is the history, as I remember it.

Student: I would call it rheumatism.

Dr. Murphy (to next student): What is the matter with the patient's ankle-joint.

Student: The patient has some infection.

Dr. Murphy: That does not mean anything to me, sir. What is the matter with the patient? That is what I want to know. I want you to look again, with your intellectual eye, into that ankle-joint and see what has occurred in it. What was the matter with that ankle-joint that gave this train of symptoms, that passed through this pathologic course, and finally resulting in the perfect joint condition which we have just been considering?

Student: It must be an arthritis or infection.

Dr. Murphy: What do you mean?

Student: There was inflammation of the synovial membrane?

Dr. Murphy: Yes, but what do you mean?

Student: The lining of the joint is inflamed. There is an exudate thrown out on the surface.

Dr. Murphy: So it was in the other.

Student: Later the ligaments undergo dissolution, and possibly the bone-ends.

Dr. Murphy: Based on what?

Student: Based on the assumption that it is an acute infection.

Dr. Murphy: I should say that your statement is based on an absence of an absolute knowledge of the real condition as far as you are concerned.

Dr. Murphy (to another student): What is the matter with the patient?

Student: As near as I can make out there was some infection in the joint itself.

Dr. Murphy: You called it rheumatism. What do you mean by that?

Student: It is a cloak for one's ignorance.

Dr. Murphy: That puts you right. Now you have a basis on which to stand. Your answer is not so obscure as the other gentleman's was. It is just like we define malaria. When a patient has malaise and we say he has malaria, the term covers a multitude of other physical sins, just the same as rheumatism does. But there are definite lesions, and these must be segregated and analyzed into a recognition of the etiologic factors, into a recognition of the tissue that is involved in the disease, into a recognition of the changes which take place in the tissue, and into a recognition of the secondary lesions that result from the infection, and into a recognition of the process of repair and its extent.

It is three years since that infection occurred. The doctor stated in reading the history that he could not locate the primary invasion. Whether you call it "arthritis" or whether you call it "rheumatism," it is a *metastatic infection* from some primary infection which occurred in some other position in the body. That is proposition number one. There is no such disease as acute idiopathic rheumatism; the inflammation in the joint is not caused by a change of the salts or other constituents of the blood. It is an infection—a secondary infection—in all the acute arthritic lesions.

When the infection is of the severe type, it is initiated with a chill, and then it is more commonly located in one, two, or three joints—rarely four. When it is more virulent it may affect a large number of joints primarily, but finally it concentrates its destructive action on an individual joint. If you aspirate the joint immediately after the manifestation of an effusion into the joint, in a large percentage of cases you will find that that fluid gives a negative result culturally, and that is why for years we concluded that this inflammation in the joint was *not of microbic* origin. That deduction is not at all justified by the *fact* that the *effusion does not contain microörganisms.*

We have only to take an analogous condition of infection in the lung which causes pleurisy. Most frequently you do not find the microörganism that produces the infection in the lung in the fluid in the pleural cavity. Why? Because it is not a surface infection; it is a subsurface infection. In a large percentage of the cases of joint infection you do not find the microorganisms in the fluid. Why? Because it is never a *primary surface* infection. Infections do not take place anywhere in the body on the surface of the synovial or serous membranes. They take place in the subendothelial layer or subsurface, in the tissues beneath. The effusion takes place as the result of the *proximity* of that infection to the endothelial surface. Infection is not on the surface of the synovial membrane, but in the tissues of the synovial membrane. It never occurs primarily in the ligaments; it never occurs primarily in the fibrous capsule. After days, and sometimes after weeks, when the multiplication of the microorganisms has taken place beneath the surface, the microorganisms begin to appear in the fluid of the joint.

The best example we can use as illustrating the delayed appearance of the microörganisms in a joint is the Neisserian infection. In the Neisserian infection the gonococcus is absent from the fluid in the joint in the enormous percentage of cases of early aspirations. Later in the disease they begin to be present, increasing from the primary findings of 7 per cent. to later findings of 80 per cent. of the cases, showing that the organism comes to the surface after the lapse of time. It is a synovial membrane infection, not a bone infection. Gonococcous infection of the subperiosteal zone occurs very rarely, but in the joint it is a synovial mural infection, and not surface infection.

The other types of infection which we so commonly see are the metastatic infections from the mouth, particularly the fauces, less frequently the tonsils, the upper respiratory tract, the intestinal tract, the urinary tract, the gall-bladder, and the genital tract, especially of the female. These joint infections are all manifestations of a *metastatic deposit* from the primary focus, and never idiopathic rheumatism.

When you get the case early, you have no difficulty at all in locating the primary focus. When you get the case late, you have great difficulty, as in this case, in locating the primary focus of infection. But you know positively that it was not tuberculosis. Why? Because tuberculosis of the joint never incapacitates a patient inside of three weeks. This lesion incapacitated the patient within a few hours. If it had been a tuberculous process, it would have continued for a long period of time, without causing any serious disturbance, the same as the lesion did in the knee. It was an acute metastatic infection, setting up a synovitis. As the tension in the joint subsided the ankle returned to its normal condition. Occasionally, however, it does not do that. When the infection is more virulent, it destroys the synovial membrane and the cartilage and ankylosis results.

The history of this case gives two beautiful pictures—one, a picture of tuberculosis, slow and insidious in its onset; the other a picture of an acute metastatic arthritis, rapid in onset, with immediate incapacity, high temperature, then all the evidences of infection rapidly subsiding, and the patient having a fully restored joint. When the metastatic infection is severe, with high temperature and associated with chill, it is not infrequently of streptococcic origin. That streptococcic infection, if there is effusion, rapidly subsides, loses its virulence, and leaves the joint undisturbed, and *aspiration* or *tapping* without drainage gives full functional restoration if resorted to *early*.

I want to show you a beautiful illustration of metastatic infection to accentuate in your minds how you must look for the primary trouble.

To Another Patient: How long is it since you had trouble with your knee?

Patient: August, 1908. I had boils on my elbow. They began on the third of August.

Dr. Murphy: What next?

Patient: I had a chill on the thirteenth.

Dr. Murphy: Then what happened?

Patient: My ankle commenced to swell.

Dr. Murphy: What next?

Patient: I had fever.

Dr. Murphy: How high was the fever?

Patient: 103.4° F. For ten days following this I was delirious and my temperature went as high as 106° F.

Dr. Murphy: When you came out of the delirium, what did you find?

Patient: My leg was swollen between the ankle and the knee. An abscess formed, and it was lanced.

Dr. Murphy: Then what happened?

Patient: My leg was lanced again and again.

Dr. Murphy: In place of having a coryza or a bronchitis or a pharyngitis, this patient had boils on her elbow, and ten days after the boils appeared she had a chill, pain in her ankle, extending to her knee. She had so much infection that she was delirious, and she woke up finally at the end of another ten days with holes in her leg and pus pouring out. That is just exactly as it occurs in pyemic conditions. She had her metastatic infection first located in the lower end of the tibia, and then it extended to the upper end of the tibia until it involved the knee and extended around the knee, producing finally a bony ankylosis of the knee and necrosis of a large zone in the tibia. She had been immediately incapacitated and has been permanently incapacitated so far as the knee-joint is concerned.

There is no question about the metastatic origin of this trouble. There is no more question about the metastatic origin of rheumatism or arthritis than there is about that. When you analyze these cases you will be surprised. In all the cases except the streptococcic variety ten, twenty, or thirty days from the origin of the primary infection metastasis takes place. In gonorrhea 90 per cent. of the metastases take place between the eighteenth and twenty-second day, showing what a systematic thing it is, how a certain evolution takes place, how it shifts to the joint. It impresses one forcibly with the fact that the primary infection is in some other position in the body. The reason you should know that is because the destruction of the joint will depend on the severity and the type and virulence of the metastatic process. You must begin the day the joint shows evidence of involvement to provide for prophylaxis against an ankylosis of the joint. That is why I wish to accentuate this. These joint infections are metastatic infections; they may rapidly become destructive, depending on the type and virulence of the infecting microörganism.

In this case there is a large osteomyelitic cavity in the bone, and as soon as that lesion is fully healed, we will do the operation for the restoration of motion in the knee. It is not a favorable case in some particulars, while it is fairly favorable in others. She has a movable patella. The patella is not involved in the disease. The infection came up through the tibia. The tibia became agglutinated to the femur, and the infectious material did not get to be free in the joint at all. It burrowed into the femur and nature coffer-dammed it. Every portion of that tibial and femoral articular surface is involved in the bony ankylosis. This is a case in which we will probably do an arthroplasty. On account of the drains in here on the side, and inability to get a soft tissue flap from the other side to interpose between the tibia and the femur, to do an arthroplasty we will take a flap from the fascia lata and fat over the trochanteric zone, detach it, and place it between the bones to prevent bony union and give her a movable joint.

POTT'S FRACTURE

Dr. Murphy (addressing student): What is the matter with this young lady?

Student: She fell down Saturday night. (This is Monday morning.)

Dr. Murphy: What happened?

Student: She slipped on the sidewalk.

Dr. Murphy: Yes, go on.

Student: She was walking on the street, fell on the right leg, and could not get up.

Dr. Murphy (to patient): Did your foot slide out or did it slide in?

Patient: It slid in.

Dr. Murphy: You sat down on it?

Patient: Yes, sir. I could not get up without assistance.

Dr. Murphy (to student): Was the foot turned in when she sat down? She could not sit on her feet unless they were turned in—you never get a Pott's fracture when the foot turns in. The foot must have turned out or everted to cause this result.

Student: She has a Pott's fracture.

Dr. Murphy: But what is the matter with her? What do you mean by Pott's fracture?

Student: Eversion of the foot in which there may be a tearing off of the internal ligament.

Dr. Murphy: Of what does a Pott's fracture consist?

Student: It is always produced by eversion of the foot.

Dr. Murphy: What happened next? What breaks when the foot turns?

Student: The internal lateral ligament is torn.

Dr. Murphy: What gives the strong support against the foot turning out? The fibula, is it not? What breaks?

Student: The fibula.

Dr. Murphy: The fibula always breaks in one direction primarily; the foot turns out, and the interosseous ligament holds the fibula to the tibia. The pressure comes on the tip, and the fibula breaks primarily. If the interosseous ligament is strong enough to hold, the fibula breaks higher up. What happens next? Then comes the break of what? The internal lateral ligament, and the tip of the internal malleolus is fractured to a greater or less degree, and as the force continues and the weight of the leg comes on the astragalus, this becomes wedged between the tibia and the fibula, forces its way upward, and splits the interosseous ligament. If the force continues still further, anterior luxation of the tibia into the notch on the top of the astragalus occurs. That is all there is to a Pott's fracture, and unless you know clearly and precisely what constitutes a Pott's fracture, you are going to pay the price by having evil results.

Let us see what a Pott's fracture consists of. First, fracture of the lower end of the fibula. Second, fracture of the tip of the internal malleolus or of a greater portion of the internal malleolus. Third, a tearing of the interosseous ligament by the wedge-shaped edge of the everted astragalus. Fourth, if the force is continued, there is luxation forward on the astragalus of the lower end of the tibia. These are the elements of a Pott's fracture. If you are going to correct and repair a Pott's fracture you must bear these in mind: First, the anterior luxation of the tibia must be reduced. Look at it! See where the head of the tibia is. It is dislocated forward.

Let us reduce it. This is the last thing that occurs in the process of these fractures. Therefore, reduce the fracture in inverse order. First, overcome the anterior luxation. Of course, the first thing is to diagnose that it is present. You know it is present by the great prominence of the anterior margin of the articular surface of the tibia. You know it is present because you are unable to swing the foot forward to a right angle with the leg. The tendo Achillis fixes it so firmly that you are unable to bring it to a right angle. The first thing, therefore, is to reduce the dislocation of the tibia. How do you reduce it? Not by trying to force the foot forward, because that locks the tibia in that notch on the upper surface of the astragalus. Increase the deformity by extending the foot, make a skidway of the upper surface of the astragalus on the posterior surface of the tibia, and then bring the foot forward; make traction and flex it, and it comes up to an acute angle with the leg. You know you have reduced the posterior luxation then.

The next step is what? To reduce the eversion of the foot. With the foot at an acute angle to the leg, evert it as far as possible, tract it downward, and *en masse* push it as far to the inner side as you possibly can. It will not go too far because the fibula will stop it from going in by the pressure on the articular surface of the tibia. Then retain it in that position, flexed on the leg, and adducted as much as possible. Why do you keep it turned in so far? Because you wish to have restoration of the interosseous ligament as close as one possibly can. Unless you restore the interosseous ligament you will have a permanent eversion of the foot, because the patient, when he starts to walk, has first a little eversion, and with the interosseous ligament, loose or torn, the edge formed by the junction of the articular surface of the astragalus and its fibular surface forms a wedge which presses up between the fibula and tibia.

When the foot is not dressed in a sufficiently adducted position, that V-shaped upper surface of the astragalus is driven up between the bones.

The method of treatment in this case will be to relax the tendo Achillis, then draw the foot forward as much as it will come, draw the toes up toward the knee, then displace the foot as far inward as it will go, and let the toes come up. The foot will be dressed in that inverted position. This brings the torn interosseous ligament into absolute contact; it keeps the foot at right angles, and the plaster-of-Paris keeps it quiet until bony union of the fibula occurs. If that does not reduce the deformity, make firm extension, put traction on the foot, throw it over, and put a cast on right away. If you will bear in mind what constitutes a Pott's fracture, you will have no trouble in treating it, remembering that the laceration of the interosseous ligament is the most important of all factors, as far as the final result is concerned. If that is restored accurately and firmly, the patient is going to walk square-footed, and the foot will be plumb with the leg.

VOLUME I *NUMBER 5*

THE
SURGICAL CLINICS
OF
JOHN B. MURPHY, M.D.
AT
MERCY HOSPITAL, CHICAGO

OCTOBER, 1912

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REMARKS ON ANESTHESIA MADE AT CLINIC

Dr. Murphy: The sister has occupied the position of anesthetist in this hospital for thirteen years. She has given anesthetics to about 15,000 to 16,000 patients, and she has not had a single fatality that could be attributed directly to the anesthetic. We have occasionally had pneumonia develop following the administration of the anesthetic, but these cases have been very rare. We believe that this excellent record is due to the extraordinary care taken by one individual who is held responsible for that work. She is, in the first place, very well informed on the subject. She reads practically all the literature that is written on anesthetics and anesthesia. She has been trained for this position. It is her life-work.

The sister sees the patient before the operation. She looks over the history to determine whether or not the report on the urine is correct. She asks the patient about the condition of the nose and throat; whether the patient has had morphin or atropin injections or just atropin. Where we want great relaxation we give morphin ($\frac{1}{6}$ grain) with the atropin ($\frac{1}{100}$ grain). Where we want merely suppression of the secretion in the upper air-passages and bronchi we give the atropin only. We are firm believers in the advantages of the atropin. We believe that it very materially lessens secretion. We believe that if we can lessen the quantity of secretion from the nose, throat, trachea, and bronchi, it materially aids in relieving any irritation that might occur lower down during the administration of the anesthetic. We have no confidence at all in the statement that if you give any of these drugs you lessen the expectorating ability of the patient on the table. A patient has no expectorating ability on the table. The idea is to keep the fluid out, and then it does not have to be expectorated. We do that as a routine, and we believe that since we have followed that plan we have had better results.

Furthermore, the sister sees practically all the cases before I get out of the hospital at noon, and if a patient is not in good shape, I get a personal report from her of that patient's condition. The next morning, or in the night, if necessary, if anything has occurred, the sister notifies the intern or me. She becomes the watch-dog of patients and she is there every day.

Contrast this plan of giving anesthetics with the methods that have been followed in other large hospitals for one-quarter of a century or more. In the first place, we find that formerly anesthetics were given to patients by men with the least experience—perhaps the youngest intern. In the second place, we have seen ether poured on by the pint. That is to say, he smothers the patient with ether. He gives five or six, or perhaps twenty, times the quantity that is necessary; he has no idea when the patient is really asleep.

I speak of these things because I was in the same position once myself, and I know that I had the greatest confidence in my ability to give anesthetics, and that confidence was increased and strongly grounded in me by my inexperience and my absolute lack of knowledge of the situation. It made me a dangerous element. I remember giving an anesthetic to a patient who was being operated on by Dr. Gunn. He said to me, "Doctor, that patient is in bad shape." I replied, "I do not think so." It took two hours to get that patient awake! He was cyanotic and nearly dead, and yet I did not have the slightest sense of danger in this case. But Dr. Gunn was kind to me. I learned my lesson that day, and the longer that patient was kept without waking, the more anxious I became. Dr. Gunn did not say very much, but he "looked a lot," and his look was enough for me. It meant something.

In managing hospitals it is also of great economic value to have a trained anesthetist. In the first place, in this institution we save more ether than it would cost to support two or three sisters. In the second place, the patients never have sore tongues following anesthesia, because the sister never permits anybody to put an instrument on the tongue. To give you an illustration: One of the visiting doctors, who was formerly an assistant of mine, was given charge of administering the anesthetic toward the end of an operation while the sister started to anesthetize the next patient. He had a forceps on the tongue of the patient. It shocked her very much to see that instrument placed on the tongue. If any of you have ever been unfortunate enough to have had a heavy hemostat put on your tongue and kept on there for twenty or forty minutes, and then experienced the discomfort for the next three or four or ten days, you know what these words mean.

In the next place, you should not only consider the great economic value of this method or system, but there is less risk to the patient, which should take precedence over everything else. We must keep that risk to the patient the dominant beacon light of our every act. The risk to the patient, or the lesser risk of pneumonia, is enormously reduced by having a special or trained anesthetist.

The variety of anesthetics you may use is very great. You may use a local anesthetic, which is less risky probably than any other. You may use nitrous oxid, which has very little risk, and which can be given in operations that last one-half hour or three-quarters of an hour, or even an hour, without accident. You may use ether alone, or ether combined with the nitrous oxid, or ether, nitrous oxid, and oxygen.

I was going to say that you may use chloroform, but I think I would put it another way: You should not use chloroform. I am not speaking as one without chloroform experience. I had ten years' service at the Alexian Brothers Hospital in this city, one of the large hospitals here, where we used nothing but chloroform, and I never saw a death from that anesthetic. However, I want to say this, that I kept up artificial respiration once for three hours and fifteen minutes, the longest three hours and fifteen minutes of my life. But the patient lived.

I remember very well one morning going into the Alexian Brothers Hospital and meeting my associate, the late Dr. Henrotin, who was coming out, looking white and pale, in place of having the thoughtful look and smile which he usually had. He looked very much perturbed. I said to him, "What is the matter, doctor?" He replied, "I just lost the only child, a boy of eight years of age, of one of my dearest friends, from chloroform." Does any one know what that means—what a direful calamity that was? How did he die? He died before he was asleep. That is just it. These patients die before they are asleep.

This recalls the unpleasant experience I had with chloroform, where I kept up artificial respiration for three hours and fifteen minutes. I went over to let water flow on my hands, turned around, and saw a change in the patient's face. In thirty seconds she was apparently dead. Her heart had stopped beating, her respiration had stopped, and she was to all intents and purposes dead. We started artificial respiration, then we resorted to dilatation of the rectum. When the rectum ceased to respond to rhythmic dilatation for six or eight minutes, her respiratory center being out of commission, we went to the upper sphincter of the sigmoid, put our thumbs away up, and ten times in a minute we would dilate to see if she would make an effort at inspiration. The heart kept on doing its work, the pallor diminished, but the minute we stopped dilating respiration ceased. We changed to the vagina, dilated that ten times in a minute, and we kept that up until she finally started in voluntary respiration. Her pulse was 200 the next day, but with care and watching closely she got well.

The danger of chloroform lies in the fact that we do not know definitely what happens. We do know that something does happen, but we do not know definitely yet what patient it is going to happen in, and when practitioners say that in this renal case or that renal case, or in this or that condition, "I use chloroform," I feel like asking the question, what do you know about that patient's ability to stand chloroform? Absolutely nothing. Ether given with oxygen, ether given with nitrous oxid, ether given alone, if administered with caution, is not any more irritating to the kidneys, we believe, than is any other anesthetic. In our experience of ten years at the Alexian Brothers Hospital we watched the kidneys of our patients very carefully,—just as carefully as we watch them here,—and we believe that the effect on the kidneys is about the same from chloroform as from ether.

In our work it is our endeavor to do the operation in the shortest possible time consistent with good work, thus making the anesthesia as short as possible. We have not used local anesthesia as we would like to do, because we have had such perfect satisfaction with ether as a general anesthetic. We believe that hernias in the aged should be operated with a local anesthetic. So that in the matter of anesthesia in this hospital, we feel fairly comfortable. We feel that the matter of anesthesia here is settled.

To the sister's mind the giving of anesthetics is to *live* in the *doing* of the work. She says: There is no set form, no code of laws, that one may adopt, for the individuality of every case is so marked that no two patients can be treated exactly alike. The service of the anesthetist must necessarily be a personal one. The physician and the scientific value of the work suffer most if she is not mistress of her art.

A few points may be mentioned merely to emphasize the fact that it is not the mere rendering unconscious of a patient that defines anesthesia, but the results that justify methods and the experience-taught lessons. Anybody can put a patient to sleep, but it takes a skilled anesthetist to know how long to keep him asleep, and what his condition under anesthesia is. Never, for one moment, should the patient be lost sight of.

The mouth and nose should be thoroughly cleansed. A small dose of morphin and atropin is given, in order to forestall any accumulation of mucus and to prevent post-operative coughing. These drugs should not be given indiscriminately, as they have a nauseating effect on some people. Good judgment must be exercised in each case, and the benefit of the patient considered first and foremost.

The anesthetist should approach her work with the composure and interest necessary to render the best possible service to her patient, who must be made to feel that she is his friend—one who is determined to do her utmost to assist him and to help him through one of his great ordeals, as patients are wont to consider operations, even though they may be slight ones. The principal remuneration of the work lies in the knowledge of the fact that the patient is in good condition after the "anesthetic stage," and that the "coming out of ether" is accompanied by as little nausea as can be expected. In all laparotomy, goiter, and brain cases *oxygen* should be given during the general anesthetic. Gas is certainly most advantageous, if a suitable simple apparatus can be obtained, by which a steady, uniform stream is assured. The patient should be kept in as normal condition as possible after operation, moderately warm. A surplus amount of heat is detrimental, as is too much cold.

Chiefly essential among the aids to establish the success of a well-given anesthetic is to labor for the confidence of the patient. If he has implicit, child-like faith in the ability of the anesthetist and her intention to be kind and watchful, it will relieve his mind of much useless worry, the absence of which is an absolute and undeniable necessity, as all will appreciate who are familiar with the details of this tremendous undertaking.

The anesthetic is begun with gas. When the patient begins to lose consciousness, the change is made from gas to ether, which is given with an Esmarch mask by the drop method. When anesthesia is complete, just sufficient ether is given to maintain sleep.

This subject is so extensive that it is impossible to give a fair outline. The work is better done than described with any true degree of accuracy, mainly because of the variety of cases and the difference in the handling of each particular case.

NEPHROLITHIASIS

HISTORY

Miss S., aged twenty-three, housemaid, came to hospital because of recurrent attacks of pain and continuous tenderness in left kidney region.

Family history negative. Had measles and whooping-cough as a child, with good recovery. Has had none of the acute infectious diseases during adult life.

Present Trouble.—When six years old she had an attack of colicky pain in the left side, followed immediately by vomiting. She knows nothing about the duration of this attack or other details. Ever since then she has had attacks of severe colicky pain in the left kidney region, radiating to the external genitals. The pains were severe enough to double the patient up, and during the past six years she has had to have morphin at each attack to control the pain. The attacks come on suddenly, last two or three hours, and disappear as suddenly. Almost immediately after the onset of the pain the patient becomes nauseated and vomits, but she states she never has had a chill or elevation of temperature, but cold perspiration comes with every attack. She has never noticed blood in her urine, but urine was darker after each attack, and several physicians who saw her during or following an attack found blood and pus in her urine. She has had no frequency of urination, but has burning at times. This burning, however, is independent of the attacks. On deep palpation the left kidney area is tender and has been tender as long as she can remember. Since the onset of this trouble the attacks have varied in frequency and severity from eight months to two days apart. Lately the attacks have been more frequent and more severe. No gastric symptoms except during the attacks. Bowels have been regular. No cough or night-sweats. No heart trouble. No loss in weight.

COMMENTS AND OPERATION

This is a classic picture. The diagram shows a stone. It should be a stone from the classic history. The skiagram must always be interpreted with an interrogation, so far as a stone is concerned.

I read a very good article in a British periodical recently which stated that in 8 per cent. of the cases where the skiagram showed positively a stone, no stone was demonstrable at the operation, and that in 6 per cent. of the cases in which there was no stone shown by the *x*-ray stones were found at the operation. So you must take the skiagram merely as a help—an adjunct—in making the diagnosis.

The diagnosis in this particular case is, of course, easy—nephrolithiasis. There has been a long period of disease, a surprisingly long period, for this girl has had renal colic since she was a child of six. When a child she was ill with these recurrent attacks of pain, nausea and vomiting, suppression of urine immediately, increase in urination afterward, the presence of pus continuously, and the presence of blood continuously now.

In addition to that, the final test that can be made at the bedside and without the aid of the microscope is first percussion of the kidney during the attack. It is one of the most positive signs and is always available. When the patient is suffering from an acute obstruction from any cause to the outlet of the ureter, or when the patient is suffering from an infarct in the kidney which increases the tension on its capsule, we can demonstrate that the lesion is in the kidney and not in the appendix, and not in the gall-bladder, by fist percussion over the spine. The value of fist percussion depends on the tension under which the contents of the renal pelvis are held during the attack. Have the patient sit on the side of the bed, or on a chair, and stoop slightly or lean over forward. Place the left hand first over the supposedly normal kidney, and bring the fist down

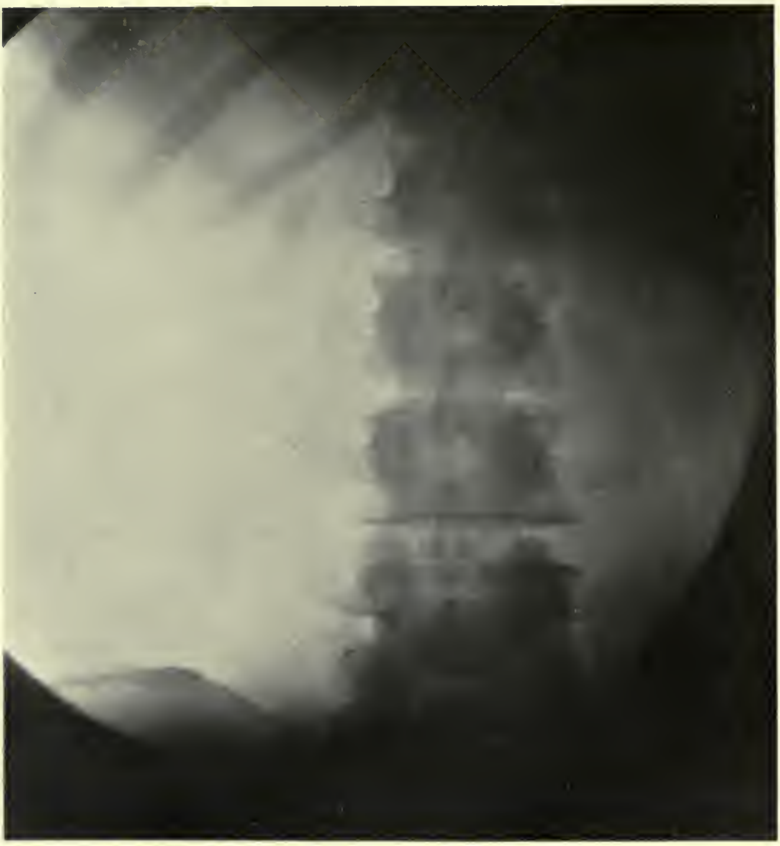

Fig. 123.—Radiogram showing stone in pelvis of kidney. Shadow about one-half inch to the right of the spinal column.

on it, striking a strong blow. There is no response; no manifestation of pain. Change the hand to the diseased side, press it firmly against the site of the kidney, and make the fist percussion, and the patient springs up suddenly and sometimes cries out. There is an immediate manifestation of pain. It is one of the most pronounced responses that I know of to physical tests of diseases inside the wall of the abdomen.

I recall the case of a lawyer on whom I made that test. He came from Gary, Indiana; he was an Irishman, and when I struck him he wheeled around and said, "Doctor, I could hardly keep from striking you—you know that hurts." I merely cite that to illustrate to you how responsive that test is. [These diagnostic methods are described and illustrated in June number.]

In this case for operation today we were unable to palpate the kidney. That means we will probably have some difficulty in bringing it down. We had the mate to this kidney the other day. The contrast in the train of symptoms is marked. That patient had recurrent attacks of pain; had diminution of the quantity of urine during the attacks, with an increase in the quantity of urine after the attack. There was pus in the urine, but she never had the typical colic described in this case. During the attack she had a typical Dietl's crisis. In that case there was an obstruction due to a valve in the ureter.

We will expose the kidney by means of the flank incision, as we did the other day. It will extend from the middle of the twelfth rib, in front of the erector spinæ, downward and forward. The length of these incisions depends entirely on the requirements of the individual case. The kidney region is quickly exposed.

I am into the fatty capsule. I will palpate for the kidney. It appears to be high up. I have it. I am freeing it from its bed and bringing it right down into the field. It does not appear to be materially enlarged. With a clinical history of a stone in the kidney for something like seventeen years one is surprised to find that the kidney is so little enlarged, particularly when we take into account the fact that the patient has had repeated attacks of colic again and again. With the upper pole free, the kidney comes readily out into the field.

The next step is to palpate for the stone, to determine whether or not she has a stone, and if she has a stone, where it is. The skiagram shows that she has a stone, and the clinical history shows she should have a stone. The skiagram shows that the stone should be well toward the pelvis of the kidney, but on careful palpation I am unable to feel a stone in the kidney. The fact that she has had repeated attacks of pain would lead one to believe that the stone should be down in the pelvis near the ureter. I have thrown the kidney forward so that I can free the renal pelvis from its fat. There is no infiltration around the kidney. There is a mass near the inner side of the pelvis that feels more like an enlarged gland than it does like a stone.

We have a rule in our surgical work on the kidney always to open the pelvis and expose the ureter and pelvis, and not endeavor to palpate through the kidney from the cortical side, but make our palpation from the other side. At the pelvis we find the first evidence of any infection. There is infiltrated tissue here. That shows that there is an infection in this zone. On opening the pelvis of the kidney I can see at once that it is dilated, and there is also infiltration of the wall of the pelvis. That is a guide to the infection that has been present here for so long a period of time. There is the stone! You remember how much that felt like a gland. It looks like a gland, but it is a stone.

Dr. Fenger advocated opening through the cortex, but this is much better. The dilated pelvis is large enough to admit my finger for a considerable distance. I will open this pelvis until I determine the position of the ureter, if I can. I have it. The ureter is patent. I will pass a catheter down into the bladder and see if we have another stone located in the ureter. The position in which this stone was located justifies exactly the clinical history.

I do not know if this catheter is in the bladder, but I will endeavor to determine. By milking the catheter or "stripping" it, if it is in the bladder, you can draw urine out of it, if the bladder contains urine. The catheter collapses after a drop or two of urine is milked out, therefore the catheter is in the bladder, but the bladder is empty.

The next step is to palpate that renal pelvis, and always do that from the pelvic side. Dr. Fenger had a great fear of opening the pelvis of the kidney, and his fear was based on his early experience. I cannot palpate any other stone.

I will close the opening in the pelvis with fine catgut. There is no occasion for doing a plastic operation, as I did on the case the other day, where we had a condition of the kidney due to a valve-like formation at the attachment of the ureter to the pelvis, also described by Fenger, but accentuated more particularly by Israel, who wrote the master work on surgery of the kidney. No other work, to my mind, has been equal to that in this particular and in its instruction, because it merely cites the cases, arranged in series, and giving the pathology. He had about 221 cases, as I recollect it now, as it is years since I read the book. The case reports in this book represent 221 accurate observations of the clinical course of the diseases of the kidney, not written in the library, and not written with the pipe. It was a record of facts, and that is what one really wants and what is going to count in the future in medical text-books, for both the practitioner and the student.

I am accurately closing this incision in the pelvis, so that there will be no primary leakage. Fenger, in one of his early cases, had a permanent fistula for which he had to remove the kidney, and that annoyed him so much that you could not get Dr. Fenger to cut into the pelvis of the kidney unless the stone and lesion were readily demonstrable. In our early work on this condition we began opening the kidney from the pelvis, and have followed that plan ever since. When I was working in St. Joseph's Hospital, nineteen years ago, I had my first important case of opening through the pelvis of the kidney and removing a stone.

There will be no drainage necessary here. If this was a large dilatation of the calices; if it were three or four times as large as now, I would put in a drain. We put in just a strand of nosophen gauze outside that line of suture and no other drain.

Let the records show that one strand of nosophen gauze is placed around the pelvis of the kidney. This nosophen gauze merely acts as a siphon drainage primarily; if urine will come out at all, it will come out along the gauze, but it does not act as a siphon for more than a short period of time. The kidney is dropped back and finds its normal position readily.

The next step is the approximation of the muscles; beginning with the location of the iliohypogastric nerve, we look for it on the inner side of the muscle, as it is here. We pick up the muscle without including it. If we include the iliohypogastric nerve in that suture, the patient will have great pain, and that accounts for so much of the pain these patients suffer following the operation, because the suture includes the nerve.

I am approximating the muscles in the regular way, otherwise I would have a hernia. Every man who has had experience in this line of work knows that hernias do occur following this operation, and they are of a very annoying type. The kidney can come out between the muscles in this position, and have recurrent luxation of it, causing great pain and great discomfort. So we approximate these muscles as accurately as we would approximate the muscles on the anterior surface of the abdomen.

In opening the pelvis of the kidney, if it is a greatly expanded pelvis,—if it is two or three times larger than we found it today,—I would make a resection of the pelvis. You will remember the case of last Saturday, in which the dilatation of the pelvis was as large as a baseball, with the long history of a Dietl's crisis. That patient had a mass in her abdomen here larger than a child's head. In that case we followed the line that we had followed in all our previous cases of this class, now amounting to 14. We did a pyeloplasty. We have made 14 complete resections of the pelvis of the kidney with detachment of the ureter and re-attachment of the ureter, with 14 recoveries and 14 good results, showing what can be done in a plastic way on the kidney pelvis.

In that case I carried the catheter down into the bladder, brought it up through the cortex of the kidney and out onto the surface. In addition to the dilatation of the pelvis, there was a dilatation of the lower third of the kidney. The secreting portion of the kidney tissue might be of the normal content, notwithstanding the great dilatation. We know that kidneys of this kind should never be removed. Why? Because you are removing practically normal kidney, and suddenly throw all the work onto the other kidney, while if the secreting tissue is absorbed gradually, or destroyed, the other kidney has gradually taken up the work and is fit to go on excreting without additional congestion and additional labor, and the patient is not affected at all by the removal.

VISITING SURGEON: Did you pass the ureteral catheter before the operation?

DR. MURPHY: No. We inspected the bladder cystoscopically, and found urine pouring out from the normal side and pus from the affected side, so that we did not need to touch the ureter. If we did not have that picture, then we would have passed a catheter. These healthy ureters should not be catheterized. I am fearful of the infections that occur from transmission of the catheters in the presence of pus.

CHOLECYSTITIS

HISTORY

MALE, aged fifty-two, married, and has three healthy children. Came to hospital because of repeated attacks of epigastric pain. Family history negative. Denies any venereal disease. Has had none of the acute infectious diseases, but has had sick headaches frequently.

Present Trouble.—Began in November, 1911, with colicky pain in the epigastrium. At the same time he states he had a sense of fulness or distention of the stomach. The pain did not radiate to the right shoulder or back, but was localized two inches above the navel, slightly to the right of the median line. Nausea came on immediately after the onset of colic, and was followed by vomiting. Patient had no chill, no fever, and was never jaundiced. The acute pain subsided in seven hours, and there was an area of tenderness over the gall-bladder for two days. Since November, 1911, he has had 9 such attacks with increasing frequency, the last two coming on within a week.

At 10 o'clock on the night of May 2d the present attack commenced, with severe pain in the epigastrium, followed by nausea. He vomited bile-colored material one hour afterward. Four hours after the onset a doctor was called, who put the patient into a hot bath and gave him some powders. This treatment had some influence on the pain, but very little. He refused a hypodermic. At this or previous attacks he had no fever, no chill, no jaundice, no clay-colored stools, no high-colored urine, no bloody urine. The attacks came on independently of meals, although he states they always followed a hearty meal.

COMMENTS AND OPERATION

DR. MURPHY: Did the patient have increase in frequency of urination or blood in the urine after the attack?

INTERN: No, doctor.

DR. MURPHY: How do you know?

INTERN: He said so.

DR. MURPHY: What was his temperature?

INTERN: His temperature was 100.6° F. up to 9 o'clock last night, coming down this morning to 96° F.

DR. MURPHY: What brought on the attack?

INTERN: The patient says he ate a hearty meal.

DR. MURPHY: Did you say that in the history? I didn't hear that. What about this attack? Let us see. This man came to my office one day this week. I examined him, but from the history was unable to arrive at a diagnosis. There were some statements that were indefinite. Following a meal he had pain, nausea, and vomiting. He never vomited that day. The pain would last six, eight, or ten days, and then disappear. He had very little temperature, very little local sensitiveness, and I could not make a positive diagnosis as to whether the trouble was in the kidney, in the appendix, or in the cholecystic tract. I told him to have a good dinner. He did, and at half-past ten that night he had an attack. It was the most severe attack he has ever had. Then he wanted to take some anodyne for it, and I told him to go through the night, that I would see him in the morning. In the morning he called his doctor, who would not give him an anodyne, and later I saw him with the condition present that I am going to mention now.

He was sensitive under the right costal arch; he could not breathe with my hand pressed beneath the arch, and on perpendicular pressure over the gall-bladder he had severe pain. He was not sensitive in any other position with the fist percussion. He had no pain; he had no blood in the urine; he had a temperature, with leukocytosis. That is the advantage of seeing a man in the attack. The practitioner who sees a patient in the attack has an advantage over the man who sees him in the interim. That is why he can avail himself of the positive signs of the presence of acute retention of bile. Remember, acute retention gives a train of symptoms valuable in making an immediate diagnosis. All in the way of physical signs disappear as soon as the acute retention is over. That is why you can tell there is an acute retention, and the principal evidence is to put pressure on any place that is under inflammatory tension. It is just like a felon. Why does a felon pain? Not because the nerves are injured, but because the inflammatory products are held under pressure. All the nerve filaments are under superior tension. It is just the same whether it be the appendix, the gall-bladder, or the pelvis of the kidney.

This man never had a temperature before, and I think he is telling the facts as far as he knows. He says he did not think he had much temperature, yet he had quite a temperature, and not only that, but he had quite a leukocytosis. This is as important as temperature, if not more so in an acute attack. The leukocyte count is corroborative of the clinical course. For many years, in all these acute conditions, we had to make a diagnosis without the leukocyte count. We consider it of very great advantage.

The time when leukocytosis is of so much value in practical work is in relation to the appendix. In a small percentage of cases of appendicitis we have, with only a very slight elevation of temperature, say, half a degree or three-quarters of a degree, or even four-fifths of a degree, a leukocyte count increased to 4000, 5000, or 6000, and in that class of cases it is valuable, because it indicates the presence of infection. The temperature and the leukocyte count are very closely related, but in a few cases, and in that few you need it very much, there is a great spread between the leukocytosis and the temperature. Occasionally the temperature will be 103° F., with a leukocyte count of only 8000 or 9000. Again, you will find less than a degree elevation of temperature and a high leukocyte count—up to 14,000 and 15,000. There are two conditions in which it is valuable to you as an aid.

This man has no history of gastric ulcer. He has no history of ever having had continued soreness. After he gets over a spell of soreness, two or three days after he gets over an attack of pain, he is apparently perfectly well again until the next spell comes on.

I recall the case of a policeman who, every two months, had to lay off duty on account of attacks of pain in his epigastrium. They would come on like colic, and were so severe that he would have to go home. We examined him, but could not make a diagnosis between the attacks. We could not do so until he got the next attack, when I could clearly outline a tumor in his epigastrium—not to the right side, but central, hard, and sensitive. I made a diagnosis of gastric ulcer, which was confirmed at the operation. This case belongs to the exceptional class, so that you must always bear that in mind in making a diagnosis of gastric ulcer, that you may have periodic pains; that they may subside and still be of the gastric ulcer type.

Remember, this man never has had any temperature, never had jaundice, and that is an important factor in all this class of cases. For a long period of time we considered jaundice as essential to the diagnosis of cholelithiasis. I have repeatedly cited my experience here and elsewhere in regard to cholelithiasis, namely, that in all the cases we have operated on and demonstrated gall-stones, in 86 per cent. of them there had never been jaundice at any stage in the history of the case. In only 14 per cent. of the cases that have come to us with gall-stones present has there been jaundice.

We make the usual incision for exposure of the gall-bladder, just below the costal arch, at the junction of the ninth rib with its cartilage, cutting through the abdominal wall, and then the peritoneum. The gall-bladder readily comes into view because of its large size.

I find here that gangrene of the gall-bladder is commencing to take place. He is in the acute stage of gangrene. There is an old infiltration present. You see the yellowness. It is a beautiful specimen, and shows that we must get at these cases early and operate promptly if we would save life. There is not a single adhesion here to protect the gall-bladder, and it is ready to break into the peritoneal cavity. When a patient has an acute attack like this man has, he is in great danger of losing his life. There is no use trying to palpate the gall-bladder for stones, as there is too much tension there. You see now the wisdom of having this man stay here in the hospital and trying to see him in an attack. He has some temperature. This gall-bladder is septic. It is brittle and under the greatest tension. I will aspirate the gall-bladder and reduce the tension. You see this looks like clear bile. The pus will commence to show when we get further down. There is thick, infected bile with blood in it. Now we have the tension off.

He never had jaundice in all these attacks. Jaundice is a symptom after the stone gets into the common duct, but it is never a symptom before, and a man may have 1000 attacks before the stone gets into the common duct.

We will determine whether there is a stone in the cystic duct,—whether there is a stone present at all,— with the type of colic he has had. One comparatively small stone I have pressed back from the cystic duct. Shreds are forming on the outside of the gall-bladder from the infection. I must examine his common duct and his pancreas. Between my middle and index-finger I have the hepatic artery. I can feel it pulsating. I know that right along that, on the under side, is the common duct, so that I carry that mass with me and know that I am going over the common-duct zone. I now palpate the head of the pancreas.

Let the record show that there is no infiltration of the head of the pancreas; that the glands at the neck of the gall-bladder are enlarged; that there is no stone in the common duct; the only stone that is demonstrable has been dislodged from the cystic duct.

Now we are ready to do a cholecystostomy and drain. We will do it in the regular way. We will not take out this gall-bladder. It is very friable, and we have to handle it with the greatest care, otherwise it will burst and that is dangerous, because it is infected. It is a little surprising to me that the intestine or omentum did not commence to cover over that necrotic zone. That man's life was unprotected. It is a common thing to have the intestine or omentum adhere around the gall-bladder, encapsulate it, and care for it, preventing the escape of infective material from the gall-bladder into the free peritoneal cavity. This illustrates a condition that does not occur very often—I will say, in three or four cases in every 100 of the acute ones, or about 3 or 4 per cent. However, they do occur.

I recall the first case of gangrene of the gall-bladder I ever saw. The patient was attacked with severe pain, followed by nausea and vomiting, elevation of temperature, and when I saw him, on the second day of the attack, his temperature was 103° F. He was very sensitive in the gall-bladder zone. We made a diagnosis of acute infection of the gall-bladder, and operated on him immediately. That was away back in the early nineties. The patient had at that time complete gangrene of the gall-bladder clear down to the cervix, but it was not separated and the peritoneum was not dead. We put in a drain. There was only one stone, and that was impacted in the cystic duct. The patient had a virulent infection.

We will now open the gall-bladder. First we insert a purse-string suture of catgut in the dome of the gall-bladder, about one-half inch from the top of the fundus, leaving the ends long. Then we insert a second similar suture, one-half inch lower down, also leaving the ends long. We surround the gall-bladder and pack this entire area with gauze, so that the infected contents will not pass into the peritoneal cavity. I will show you that it is gangrenous. You will see that the mucosa is gangrenous; that it is not the color of the gall-bladder that shows, but it is the gangrenous mucosa. There are a number of stones present, although I felt only one. There are four stones. What a wicked thing that was in such a short period of time.

I am making this opening a little larger for my rubber drainage-tube. Gangrene of the gall-bladder is present already, and it shows how quickly this gangrene takes place, but this is exactly in consonance with our knowledge of gangrene that occurs in connection with appendicitis.

We will turn the mucosa in. We will make an inversion of that cut edge of the gall-bladder, so that any bleeding that may take place will do so into the gall-bladder. I will fix the gall-bladder at this point to the tube, so that the tube and gall-bladder cannot separate, and before I draw the second row of stitches taut, I will push the tube down into the gall-bladder, inverting or burying the cut edge in the gall-bladder, making an entropium.

VISITING PHYSICIAN: Is that silk you are using, Dr. Murphy?

DR. MURPHY: No, it is catgut.

We will put a second tube into the peritoneal cavity, outside of the gall-bladder. I will stitch that tube to the gall-bladder tube. The needle, in passing through this rubber tube, does not make a hole that will leak; the rubber tube contracts onto the needle, so that there will be no leakage from the needle-hole. This is an extracholecystic drain in the fossa, because that fluid is septic.

We will now commence to make the usual ectropium of the edge of the peritoneum, using catgut. That prevents the formation of adhesions within the abdomen.

VISITING PHYSICIAN: Do you sew the gall-bladder to the parietal peritoneum?

DR. MURPHY: No, not at all. When we started to do gall-bladder surgery there had been only 13 cholecystectomies performed, I believe, up to that time. Then we fixed the gall-bladder to the parietal peritoneum. We were afraid to drop it back as it is dropped back here. Then, as gall-bladder surgery evolved, we fixed the gall-bladder to the peritoneum low down. Originally we fixed it to the skin and had a cholecystic sinus form. As the evolution of this work went on we came to the plan that I think was first introduced by Mayo, that is, of putting a tube in the gall-bladder, suturing it, making an ectropium of the cut edge, and then putting in a second line of sutures, as we did here, and drop the gall-bladder back into the peritoneal cavity. Remember, the tube has only two holes in it—one at the end and the other at the side, near the end. The cut edge of the gall-bladder is turned in and bound around it firmly. The gall-bladder is then dropped down into the peritoneal cavity. This tube will drain everything from the gall-bladder. I put in a second drainage-tube because there is a septic condition present here. It will drain the duodenohepatic fossa, taking care of any material that escapes from the gall-bladder.

Visiting Physician: How long do you leave the tube in?

Dr. Murphy: It will be left in twelve or fifteen days in this case, because a portion of the mucosa will slough and come out through the hole made by the tube. We do not disturb it at all for twelve or fifteen days, and then take it out and inspect the gall-bladder. At the end of five or six hours nature will have encapsulated the tube and shut it off from the peritoneal cavity. At the end of fifteen days you have a connective-tissue canal formed down to the gall-bladder, where the mucosa is showing up on the inner side. There was free fluid in the peritoneal cavity in this case.

Let the record show that the gall-bladder was greatly enlarged; that it protruded well below the costal arch; that it was gangrenous over an area as large as a nickel; that the neighborhood of that was congested, and looked more like liver tissue than like gall-bladder surface; that there were no adhesions whatever to the gall-bladder at the position where it was going to perforate, and if it had perforated, it would have done so directly into the free peritoneal cavity.

On palpation of the gall-bladder a small stone was found down in the cystic duct; this was displaced out of the cystic duct. Three other stones were removed. The patient never passed a stone through the common duct; had never had a jaundice at any time in his attacks. When these patients pass the original stone through the common duct, jaundice uniformly occurs, and sometimes even when the stones are small, because it produces trauma and edema of the mucosa around the stone, and thus a comparatively small stone occludes the common duct and causes a typical jaundice. If the stone has been in a considerable time, the jaundice may entirely subside, and one often finds a stone in the common duct where the patient has not had jaundice for years, but one rarely ever finds a stone in the common duct where a patient has not had a history of icterus at some time in the course of the attacks.

Continue with the record. Further examination and palpation of the common duct revealed no stone. There was no infiltration of the head of the pancreas. The condition of the gall-bladder shows that there was an acute gangrenous infection of an old infiltrated gall-bladder, because the surface of the gall-bladder could not change to the condition it was in in one single attack of short duration, namely, night before last. This patient is getting his reward, because he did what he was asked to do, which accounts for the fact that he will not have a septic gall-bladder rupture into the free peritoneal cavity at 8 or 10 o'clock to-night. It takes the gangrenous portion of the gall-bladder a little longer time to separate than in the case of the appendix. The gangrenous portion of the appendix can separate as early as thirty hours. The gangrenous gall-bladder does not separate as early as that because it has more fibrous tissue in it, and it has not so rich a circulation.

The stomach was free from adhesions, as was also the duodenum. There was considerable free fluid in the peritoneal cavity. It was not very cloudy. With a gangrene of the appendix as severe as that and approximating the surface as near as that did there would be a milky fluid or what we call a purulent fluid in the peritoneal cavity. That purulent fluid, before perforation, is not pus. It is a defensive fluid, and not an offensive fluid, namely, it is lymph filled with polymorphonuclear leukocytes to take up the microörganisms when they come in.

The abdominal wall will be closed with a figure-of-8 stitch. Horsehair will be used to approximate the cut skin edges.

As to the subsequent course of this case: Will we have superficial infection here from the pus and bile that came from the gall-bladder? We do not know. What is the rule? The rule is that we do not. The rule is, with a more purulent material escaping than we have now primary union of the abdominal wall occurs down to the tube, the bile and pus both passing into the wound. Why that occurs we do not know.

The same thing occurs where you have an offensive, rotten material escaping from the surface during the opening of a peri-appendiceal abscess. The infection more frequently produces necrosis of the tissue in the neighborhood of the tube, but not infrequently, with the most foul material, you get primary union of the abdominal wall where the wound has been smeared with pus. Even with all our care and precautions, occasionally, in preparing the wound and no pus in the field, we find difficulty in keeping it aseptic. So, you see, there are some things about sepsis that have to be cleared up, and, notwithstanding the fact that we sit back and fold our arms and seem satisfied, we have a lot of things to learn why one case does badly and why another does not.

We have learned some things in connection with arteriosclerosis cases. These patients stand infection so very badly that when one has anything like a severe infection in a patient with arteriosclerosis, that patient is likely to die.

We have had in the last few years some striking cases of that in this hospital. Four or five weeks ago a patient came in with hemorrhoids. Following that he had a perihemorrhoidal abscess in the perineum. He was an arteriosclerotic. He began to get an infection, and on Monday, after having got off the train in the morning coming from St. Paul, he went to his home. Later in the day he came for treatment, and in the afternoon, after 3 or 4 o'clock, we saw him. His scrotum at that time was infiltrated and black. His perineum was likewise infiltrated and black, and he had a subcutaneous infiltration extending up to the level of the umbilicus, so that when we cut it open along both sides parallel with Poupart's ligament, we found an emphysematous gangrene extending up to the level of the umbilicus.

Another case similar to that was a preputial abrasion. The man took a walk of a mile and a half on a warm spring morning and got an abrasion of the prepuce. This occurred on Friday. On Sunday evening he had a mild chill; on Monday, a severe chill; and on Tuesday he had another chill. Then he told a doctor about "a little inconvenience" he had. He was treated for malaria, which means nothing at all at that season of the year,—in March, I believe,—because the mosquitos were not alive in the district in which he was living. That statement was undoubtedly made by the doctor to cover an error in diagnosis or his inability to make a diagnosis. He had a second chill on Tuesday.

I saw him on Friday, and he had complete gangrene of the scrotum, of the corpus spongiosum, and the gangrene extended up, as it did in the other case, to the level of the umbilicus. He had had arteriosclerosis, with angina pectoris, for twelve years, to my knowledge, and had attacks of angina pectoris at night for twelve years preceding, showing the virulence of the infection or the absence of resistance in the arteriosclerosis cases.

When these patients have a mild infection, it is much more serious than a similar infection in younger people, and hence one has to do the work with great caution, and particularly in loose cellular tissue of that variety.

I will ask the intern to reread the history of this case, so that we can make some comments on it. [Here the intern reread the history.]

Dr. Murphy: You can see why we put it cholecystitis in place of cholelithiasis, because he had pain which was not a typical colic. He had the soreness and resistance to percussion and to the "hook" test under his costal arch, and he has leukocytosis and elevation of temperature. This shows you the point we have accentuated so long, that the cases of infection in the gall-bladder have manifestations in the way of elevation of temperature, varying with the location of the infection or the location of the foreign body. When it is in the gall-bladder, you rarely have a temperature which exceeds 101° F. When it is in the cystic duct and there is sepsis present, you frequently have a temperature running to 103°, 105½°, or 106° F. When it is in the common duct, with the stone retained there, and there is infection present, the patient has chills and fever, the same as he has when it is in the cystic duct, with high temperature and icterus added to it. The last is the most dangerous class of cases we have to deal with, because with icterus and sepsis both present, and with a stone impacted in the common duct,—acutely impacted in the common duct,—there is danger of an ascending cholangitis occurring, going clear up into the small bile-ducts, from which the patient can and often does die, with symptoms of what was called icteric necrosis, with retention of bile and small necrotic foci throughout the liver.

In the other variety, where that does not take an ascending course, intense sepsis is present in addition to the cholemia. That is the class of cases that gave Kehr so colossal a mortality, but that is the class of cases in which you would not operate at this time. If that man had an icterus, with a temperature of 103° to 105° F., all coming on within the last few hours, and you operated on him, he would die, as a rule. I make that statement because it is based on Kehr's statement that he had 97 per cent. of these cases terminate fatally. We do not do operations that have a chance of life of only 3 per cent. It is not good surgery. A larger percentage of these patients will live, get necrosis of the common duct, get pericholelithic abscess, get an abscess in the upper abdomen, and still live, with a subsequent drainage, in more than 3 per cent. of the cases when interfered with in the acute stage.

Here in this case it was different. There was no jaundice and had not been. Here we had all the conditions showing retention of infection under pressure, with only a little absorption. When there is this great pressure in the gall-bladder, why is it you do not have as high a temperature as you do when there is a little stone in the cystic duct, or in the common duct with infection present? Because the fundus of every viscus has but few lymphatics. The fundus of the gall-bladder, the fundus of the urinary bladder, and the fundus of the stomach have but few lymphatics. They do not get the sepsis from there. The absorption occurs only where there is a rich lymphatic supply, beginning at the infundibulum of the cystic duct and extending down to the ampulla of Vater. That rich lymph supply admits of the rapid absorption of septic material, chills and fever, with recurrence, resembling the recurrence of chills and fever of malaria.

When I was a student, acute malignant icteric malaria was regularly diagnosed as a stone in the common duct, with chills and fever that came on every second or third day, like the chills and fever of malaria, and the first man in this country, to my knowledge, to elucidate that subject was Dr. William E. Quine, of this city. He reported four postmortem examinations in which he demonstrated stone in the common duct, with the typical classic condition reported, and as described in Trousseau's Internal Medicine, as malignant malaria, due to what? Due to the sepsis from a stone impacted in the common duct.

GASTRODUODENAL ULCER—GASTROENTEROSTOMY

HISTORY

Male, aged forty-one, farmer. Drinks moderately. Smoked to excess up to four months ago; since then has been chewing. Denies lues or gonorrhea. Had pneumonia when a child. Right clavicle fractured twenty years ago.

Present Trouble.—Began five years ago. Once a year, during the first three years, he had attacks of gastric pain, lasting five or six weeks. The fourth year the attack lasted three months, and this last year it lasted seven months. Began May, 1911, and lasted until December, 1911. Pain came on from thirty minutes to four hours after eating, generally in from two to three hours. It lasts until he vomits or until the next meal, when eating relieves it. It is in the epigastrium; does not radiate. He wakes up sometimes at 12 P. M. or 1 A. M. Solid food causes more distress than liquids. Soda gives some relief. Nauseated four hours after eating, and only vomits when he gags himself. Vomitus is sour; contains no blood, but has contained food eaten twelve hours before. Belches a great deal of gas. Has a regurgitation of sour fluid. Not tender over his stomach. Never fainted. Bowels constipated; stools never black, except when taking medicine. No headaches. Has lost 28 pounds in the past eight months. Since December, 1911, he has not had pain, but has heavy feeling in stomach and it is distended from thirty minutes to two hours after eating. This lasts until the next meal, when he is relieved for from thirty minutes to two hours after eating. Nauseated twenty minutes to one hour after eating. Never vomits unless he forces himself to do so. Skin had a yellow tinge two months ago. Heart and lungs negative.

Examination.—No tenderness in gastric area or elsewhere in abdomen. Gall-bladder is absolutely free from tenderness on deep percussion and finger pressure. No rigidity or tenderness of spinal column. No signs of locomotor ataxia. Left iris reacts only slightly, due to trauma to eye fourteen years ago, with adhesions. There is an area of tenderness on deep percussion which can be found with difficulty at a point corresponding to the pylorus. It is probably a saddle ulcer at pylorus.

COMMENTS AND OPERATION

Dr. Murphy: The history extends back over a period of five years, with how many attacks?

The Intern: Seven attacks, doctor.

Dr. Murphy: What were the abdominal symptoms?

The Intern: Pain two or three hours after eating.

Dr. Murphy: Lasting how long?

The Intern: Until the next meal.

Dr. Murphy: Relieved by what?

The Intern: By eating and vomiting when he gagged himself.

Dr. Murphy: Relieved by emptying his stomach or eating. What about the night disturbance?

The Intern: He would wake up about 12 midnight or 1 A. M., and turning on the left side would give him some relief.

Dr. Murphy: Turning on the left side would give him relief?

The Intern: Yes, doctor.

Dr. Murphy: What else? The first attacks came on what time of the year?

The Intern: In the spring.

Dr. Murphy: The first attacks came on in the spring and fall. The rule is they always come on either in the spring or fall, rarely ever in midwinter or in midsummer. That fits exactly with the common course of these lesions. Now what else? What other symptom have you not accentuated in your history?

The Intern: Feces examination showed blood chemically, and stomach-contents showed blood.

Dr. Murphy: But he never had a severe vomiting of blood;

never had a hematemesis, and he never had a melena; never had a large quantity of blood discharged from his bowel.

The Intern: No, doctor.

Dr. Murphy: What other symptom have you not mentioned with force? What other symptom do these patients have? the symptom that is so characteristic of these lesions?

The Intern: Localized pain. He had that, doctor.

Dr. Murphy: Remember, the train of symptoms are pain relieved by taking food. If the ulcer is inside the pyloric zone, pain comes on earlier than when the ulcer is outside the pyloric zone. If the ulcer is outside the pylorus, in the first portion of the duodenum, pain occurs two and a half to four hours after eating. When the ulcer is inside the pylorus, you have pain increasing up to the intake of food; when the ulcer is outside of the pylorus, you have pain relieved by the intake of food. He says this patient had to get up at night about 12 or 1 o'clock, which is a common thing in duodenal ulcer, and if he took sodium bicarbonate or a glass of milk, or even a glass of water, he was temporarily relieved. We have all believed that food relief is due to the fact that the food stops coming through the pylorus. We do not know that that is true, and it does not matter.

The next symptom of importance is the appearance of blood in the vomitus and in the stools, either microscopically or macroscopically. With a severe duodenal ulcer the patient may vomit the blood as well as pass it in the stools.

These patients get up in the morning feeling as well as usual and go to work as usual; suddenly they commence to feel discomfort in the abdomen; feel faint, do faint, and then vomit a large quantity of blood, with loose bowels for two and a half to three hours. So classic is that history that it guides one at once to the diagnosis, and that is why the doctor mentions that this patient never fainted and has no history of a severe hemorrhage.

Then we go on to the next symptom—the loss of weight. The loss of weight is usually not great. You will find that these patients with ulcers for weeks and months and years maintain about their regular weight. This man has lost 28 pounds in six months. We charge up part of that loss to the restricted diet he had been on, but the restricted diet does not begin to take off 28 pounds. There is something else wrong.

Next is the symptom of gas. It is one of the conspicuous symptoms of ulcer, coming one, one and a half, sometimes two hours after the intake of food, and it comes in large quantities; the patient becomes distended. Following the distention the patients will tell you that if they get up large quantities of gas they are relieved. It takes the tension off the ulcer, that is why the patient is relieved.

Pain in the duodenal type of ulcer, as a rule, is never acute. Pain in the gastric type of ulcer can be very acute and resembles a colic, but in the duodenal type it is more a sense of pressure and disturbance in the epigastrium than a real acute pain. One of the other conditions associated with the train of symptoms is pain. Pain after the intake of food, increased by the further intake of food, and not relieved by the intake of food, and continues for two and a half and three hours and then is most pronounced two and a half or three hours after the intake of food, with sensitiveness in the epigastrium, is the pain of gall-stones. You remember the case we operated in our last clinic. The woman's symptoms were associated with food intake coming on two and a half or three hours after meals. She had attacks of pain, not severe enough for colic, but a severe pain and distress after meals. On operation we found an overdistended gall-bladder of very large size, with grumous bile. She had gangrene of the gall-bladder. That was the appalling thing, and with symptoms so mild. The gall-bladder was absolutely gangrenous, without a single adhesion or a thing to prevent it from rupturing into the free peritoneal cavity. That accentuates the relationship between the gastric symptoms and the overdistended gall-bladder, rather than gall-stones.

I will make the usual incision through the rectus muscle. We lift up the rectus sheath on the inner side, and displace all the muscle-fibers upward. Lifting that up in this way we go to the linea alba and simply displace these fibers. I am down on the deep rectus sheath. When I suture that sheath the muscle will fall back again to the central position. I always cut the peritoneum just below where it is grasped with two tissue forceps, not between the forceps. Always cut on this slanting line. As the doctor pulls up you will see that when the abdomen is opened the omentum and stomach slip out of the field so that they cannot be cut. There is the stomach now in the field. I put on these towels to protect the abdominal contents from coming in contact with the abdominal wall. Then we lift up the bowel. The ulcer is plainly to be seen. It is right at the pylorus. See the white line! There is where the ulcer endeavored to perforate. I will see whether it goes out into the duodenum. No, the ulcer is on the pyloric side. The patient came to us against the advice of the medical man. He came just in time. The ulcer would soon have perforated. There is the result of an ulcer of five years' duration. The spot where it has endeavored to come through is a hard mass.

We must first take care of that perforation and sew over it the omentum. I sew it over the indurated area, so that it will be protected and heal. The omentum will seal all that place and prevent leakage during the early hours after the operative procedure. I am just putting on an omental patch there. Dr. N. Senn is the man who worked out that patch idea originally, and all the patchwork is based on Senn's original experiment. I am going away beyond the line of induration to catch up my patch. This is the best class of case for operation. The ulcer is big, it is near the pylorus, and interferes with the outline of food.

The next step is to do a posterior gastro-enterostomy. There is the ligament of Treitz, and there is the first portion of the jejunum. The ligament that is attached to the first portion of the jejunum is visible.

The next step is to expose the posterior wall of the stomach and open the lesser peritoneal cavity. I will make that opening large enough so as to bring the posterior wall of the stomach into the field, and I want to bring it out as near to the pyloric zone as I can. I will lift the posterior portion of the stomach out of the peritoneal cavity, and that will close the lesser peritoneal cavity so that the intestine cannot find admission to the lesser peritoneal cavity following the operation and become strangulated. This is the first step—to see that that accident cannot occur, and that that line of union is not going to be hazarded by anything intervening. That portion of the stomach cannot any longer recede. There is the portion of the stomach to which I am going to make my attachment, not transversely, but in a downward direction with the food current.

Now we will empty the jejunum downward and put on the intestinal clamp in the regular way.

I am getting ready for the combined operation of gastro-enterostomy, a different operation from that we have been in the habit of doing. It is the combined operation of button and suture. I will show you how easily that can be done.

The greatest danger in a gastro-enterostomy is hemorrhage. Everything here is protected, however. We use two straight needles threaded with Pagenstecher linen to make the intestinal suture. We use the Hartmann holder for steadying the button, after having put in the stitch. The suturing is done by passing these long straight intestinal needles parallel to each other through the bowel, and then the bowel is incised between the needles. We are then ready to insert the button. Half of an oblong button is inserted into jejunum and stomach respectively.

The next is to take up the stomach. We pull out the stomach and put the clamp on it and get it up as much as we need for the purpose of inserting the button. We insert the needles here just as we did in the case of the intestine; take up all the coats of the bowel in the stitch. It is the same proposition as before—divide the stomach between these two needles. After the two halves of the button are in place, we pull the suture taut so that both stomach and bowel are held firmly against the button. Now we are ready to make the approximation. We press the two halves of the button together tightly, catching stomach and bowel between them, so that there will be no leakage. Then we take off the clamps. The approximation is perfect.

Now we commence to put in the tacking stitches on the side. I will show you how quickly you can run a stitch all around the line of union and insure against hemorrhage and against leakage. Usually we just put in a single tacking stitch here and there all the way around. In place of that we will put in a continuous stitch around the circumference of the approximation. You can do this operation in any combination you desire.

While in Europe a year or so ago we saw a great many gastro-enterostomies done, but no two men were doing gastro-enterostomies in the same way; at the same time all were getting good results. With the curved needle used by Moynihan, even though it is a large needle, you can get in almost any position in this region that you desire.

What will be the subsequent course of this case? It should be that he will have a drainage here; his food in the future will not pass over the ulcerated zone, but it will be short-circuited. Now we drop that back into the abdomen. It falls back into the normal relationship of his jejunum and stomach, except that in the future there will be no interposing mesocolon between his stomach and his jejunum.

The next thing is to suture the peritoneum by making an ectropium of the serosa. You can see how I make that ectropium of the serosa, by simply passing the needle all of the time inside the edge. That was the combined suture and button operation, with the continuous suture. The abdominal wall is closed in the usual manner—with deep and superficial combined figure-of-8 catgut and horsehair skin sutures. The wound is dusted well with bismuth subiodid powder and sealed with collodion. The usual abdominal dressing is put on. In this case we also sutured separately the posterior and anterior rectus sheaths.

Let us go over the history again, while we are looking backward at our diagnosis, after we have had "the proof of the pudding," and see how it fits with the symptoms, how it fits the symptoms of a gastric ulcer when the ulcer is of long standing.

He had evidence of hyperacidity, which is so characteristic of ulcer in the pyloric zone—more common, I would say, when the ulcer is outside the pylorus than when it is inside. I think it can fairly be translated in this way: that the gastric hyperacidity, as it is commonly called, is caused by ulcer in over 80 per cent.

of the cases. Hyperacidity is to be interpreted differently from what it was before. We are in the next five years going to change the word hyperacidity to ulcer in the neighborhood of the pylorus. The hyperacidity is merely one of the symptoms. So commonly is the ulcer associated with all the conditions known as hyperacidity, and so commonly is hyperacidity made as a diagnosis, while hyperacidity is a symptom and not a condition at all. That is the proper way. There are many conditions that cause hyperacidity, but the most common is ulcer.

This man had his pain two or two and a half hours after his meals. That is exactly right for the location of that ulcer, and exactly the same as if the ulcer were outside the pylorus, in the duodenum. We have endeavored to accentuate the fact that the difference between these two conditions is not sufficient to make a differentiation.

This is a very beautiful illustration of what we find in this class of cases. These ulcers do not heal under medical treatment in the large percentage of cases. In about 70 per cent. of the cases we get permanent healing with gastric drainage of the type I have performed. In about 15 to 20 per cent. we get a great improvement in the condition. In the other 10 or 15 percentage we have recurrence of the ulcer, but that is the exception.

That ulcer was exceptionally large. It was very thick, and that is just the sequence of five years' effort on the part of nature to repair the lesion, and it does there just what it does in so many positions in the body. Wherever you have chronic ulcer you have constant coffer-damming. You saw the anemia of the central zone—the white patch, that is where the perforation occurs.

The button will slough out as healing takes place, and will be passed with the feces. Sometimes it passes soon after the operation, but occasionally it is not passed until late. In a recent case it was passed on the eighty-first day. [In this case the button was passed on the eighteenth day.—Ed.]

APPENDICEAL ABSCESS

HISTORY

This girl is a student, fifteen years old. She entered Mercy Hospital because of pain and excessive tenderness over the entire right iliac region.

Present Trouble.—The present trouble began two weeks ago. She had a headache and sore throat, and insists that she felt feverish, but had no pain in any part of her body. That was on Thursday. The next day she stayed in bed and felt feverish and at times chilly. Saturday she remained in bed, also on Sunday, when she vomited twice. On this day she experienced severe cramp-like pains about the center of the abdomen. These continued until Wednesday, when she had pain for the first time in the right iliac region. Her temperature on this day was 100° F. She has been in bed ever since and has had a temperature continuously. A doctor who saw her late Friday elicited tenderness in the right iliac region. Leukocyte count, 12,000.

COMMENTS AND OPERATION

Dr. Murphy: How long after she had the initial symptoms did she vomit?

The Intern: The trouble began on Thursday.

Dr. Murphy: The trouble began on Thursday? When did the pain begin?

The Intern: She began with symptoms of the grip on Thursday.

Dr. Murphy: I want to bring that point out because of the class of cases which we had in Chicago this last winter. It is a new type of cases to me and to the whole medical profession. The epidemic was most pronounced in Chicago, next in severity in Philadelphia. There have been a number of cases of peritonitis from disease of the appendix—a slight edema and swelling of the appendix, congestion of the mucosa; an extensive reddening of the peritoneum. It all began with this type of septic angina. The patient had a sore throat and headache and all the evidences of grip. This is what it was called in the early part of the season, but we learned later that it was a peculiar and apparently specific type of infection due to a coccus resembling the diplococcus of pneumonia, but it was not the diplococcus of pneumonia, so far as we were able to learn. I recall two families where the disease affected every member of the family; each one in each family had a typical attack of infection of the peritoneum, as indicated in this case.

In all these years I have talked to you about appendicitis pure and simple. I have told you that appendicitis always began with pain, nausea, and vomiting. Here is a type of case in which the trouble did not start in the appendix. It began as an angina, and then the appendicitis came on with the regular cycle of symptoms.

We lost one of our patients. I operated and found that condition of the appendix—that peritonitis. She recovered from the peritonitis and finally developed a pneumonia. She developed tympany, which is so commonly associated with pneumonia.

We had another case that began in practically the same way. The patient had recurrent attacks of appendicitis and came into the hospital on the third or fourth day of the appendiceal trouble. It was a clean-cut case. The late Dr. Henry Lyman recognized in a series of cases that a pharyngitis came on first—a postnasal condition—and then an attack of appendicitis. He believed that it was "rheumatism" of the appendix. Dr. Lyman recognized the sore throat in connection with the appendicitis. I remember the first case I saw in consultation with him, because I was so positive on one side and he was equally positive on the other, and we were both right. The patient was a child. He had pneumonia when he came in with his appendicitis. We did not operate on him. If you operate for appendicitis in the presence of pneumonia you usually have to sign a death certificate afterward as the reward for your trouble. We tided him along until his pneumonia disappeared; then we operated.

This patient has been operated on before, which was not mentioned in the history. A drain was inserted. I am going on in the line of the first incision, opening the abdomen in the usual way. The omentum is adherent. We will separate that adhesion.

There is the cicatricial tissue all along the line of the drainage-tube. All the intestine is adherent at this point. We must carefully liberate these adhesions before we will be able to expose the appendix.

That done, the caput coli rolls up into the field. We have the third degree of obstruction around the base of the appendix, and there we will have organic adhesions. There is the ileum. There are the adhesions from the ileum down onto the base of the appendix. I will free these adhesions and see if we have any part of that appendix left. I can see where the abscess formed. There is gray granulation tissue around the site of the abscess. That is the result of the infection. The gray mass is the degenerating product of infection getting ready for absorption. I can feel the thickened mass where this abscess burrowed down into the neighborhood of the great mesentery.

I will free this mesenteric adhesion. These adhesions are not organic; they are not fixed. Here is the ileum and the ileocecal valve, and here is where the appendix is normally located. I am trying to feel anything that I might recognize as the stump of the appendix. I cannot see or feel anything like an appendix attached to the caput coli where the appendix ought to be. I will free these adhesions further back and see whether the distal end of the appendix is here or not. The proximal end appears to have sloughed off from the bowel. I can feel something like an appendix. Here it is, and I am rather fortunate to find it. There is only the tip left. The appendix has sloughed. We will free that from its mesentery and I will be able to bring it up. All the distal portion has sloughed off. This girl would probably have had recurrent attacks of inflammation in the stump, but she never could have had a typical appendicitis. We advised her to go home, but she felt if she went home the attack would come on again, and she decided to have the operation done at this time.

We will put in the puckering-string suture in the usual way and ligate the vessel of the mesentery.

We will conclude the operation in the regular way and remove the remnant of the appendix.

This case illustrates so many points in connection with an operation at this stage of the disease, and particularly the process of absorption of the adhesions. Now we put in the regular Lembert stitch and cover up all that line of union. The danger of infection in this operation is very much less at this stage of the disease.

We will close the peritoneum with a continuous catgut stitch by everting the edges, and then suture the abdominal muscles carefully so as to forestall the possibility of a ventral hernia occurring later. Figure-of-8 sutures close the fascial incision; and horsehair approximates the skin-edges. We do not put in a dressing. A gauze collodion dressing seals the wound, thus guarding against infection from without. [The patient made an uneventful recovery.—Ed.]

COLONIC ADHESIONS SIMULATING RECURRENT APPENDICITIS

HISTORY

FEMALE, teacher, twenty-one years old, entered Mercy Hospital because of recurring attacks of pain in right iliac region. Her menses have always been regular, and she has always been free from pain. A slight leukorrhea has been present for the past eighteen months. She has never been married and never missed a menstrual period. Her life has been free from serious illnesses. She never has had typhoid or other intestinal disorders.

The first abdominal pain this girl ever experienced was in the summer of 1906. It was in the right iliac region. It never became severe and caused her inconvenience only for one day. In the fall of 1906, three months after the first attack of pain, she was seized by a sharp, colicky pain in the right side of the lower abdomen. It was quite severe, but not sufficient to cause her to leave her class-room, and persisted only for one day. During 1907–08–09 she remembers having had attacks of similar pain at intervals of from one week to two months. These never kept her out of school, and the pain always passed off after one or sometimes two days. She never became nauseated, never vomited or had a fever at these times, as far as she remembers. Between attacks her health was perfect. She never had eructations of gas or a heavy sensation, as of fulness, in epigastrium after eating.

Since 1909 the pain has been more severe. It has recurred nearly every two or three weeks. Seven times she has been kept in bed for three or four days at a time with very severe pain, always at McBurney's point, but often radiating to the center of the abdomen or to the right lumbar region of the back.

At these attacks she has always been nauseated and had emesis and has often felt feverish.

The present attack began Saturday, as usual, with severe, colicky pains at McBurney's point. She went to bed immediately and has stayed there ever since. Two hours later she vomited. She had no chill nor has she ever had one. She is sure she had a fever. She noticed, as she has often done during these attacks, that the muscles of the right lower abdomen seemed rigid and that a swelling the size of an egg rose up at the site of the pain. This was directly over McBurney's point. The pain has subsided and recurs only if she turns quickly in bed. A tenderness still exists. Temperature on entering hospital, 99.8° F. She has a slight cough. Heart and lungs normal. Slight leukocytosis.

COMMENTS AND OPERATION

DR. MURPHY: What about those last attacks, doctor?

THE INTERN: She was nauseated.

DR. MURPHY: She was incapacitated for three or four days?

THE INTERN: Yes.

DR. MURPHY: Was her temperature taken at any time?

THE INTERN: No, doctor. [Reading further.]

DR. MURPHY: Swelling comes on only at the site of the pain? When does it come on?

THE INTERN: After each attack.

DR. MURPHY: After each attack. What do you mean? "After" is a very indefinite time.

THE INTERN: This is not my history, doctor.

DR. MURPHY: It does not make any difference whose history it is. Does she get that pain and then does she get elevation of temperature, nausea, and vomiting? Does all that occur within a few days? When there are fixation and binding of the ascending colon by these pericolonic bands, and in these cases this produces temporary obstruction and distention, with enormously dilated caput coli, hypertrophy of the caput coli, with sensitiveness during the period the tension is on, then rapidly subsides and disappears until another attack comes on. So classic is that condition that you can make a diagnosis on this circumscribed condition or on this sensitiveness to pressure and on the fact that when the patient commences to get well he will tell you of a rumbling of gas, an explosion, and the attack is over. When you analyze these cases you will be surprised how accurately they describe that condition.

That is not recurrent appendicitis at all; it is recurrent retention and distention of the caput coli and is classic, and many of these patients are operated on as appendiceal cases, with no attention paid as to the facts, with the result that the patient comes back again and again, and goes from one doctor to another seeking relief. The kidney is tied up, the appendix taken out, a tube taken out and probably an ovary, and all the time the diagnosis is recurrent appendicitis. It is only when we make a careful analysis of these cases that we can make a differential diagnosis. One operation after another is done on the right side of the abdomen, and, as the Mayos would say, all the movable organs are fixed and all the fixed organs are made movable, and finally all those that can be removed are removed, and still the patient is not relieved of the pain and other symptoms.

DR. MURPHY (To intern): What was the leukocyte count when she came in, doctor?

THE INTERN: 5800; the count yesterday was 9000.

DR. MURPHY: Was a tuberculin test made?

THE INTERN: No, doctor.

DR. MURPHY: It should have been made. Why? Because she had a leukopenia when she came in. Either that count is incorrect or there is something wrong somewhere. It does not fit. She had no soreness when she came here. She came with an acute exacerbation the day before yesterday, and then had a leukocyte count of 9000 yesterday; you see how one little feature puts things all out of gear. She had an acute attack, and with this attack she had a leukocyte count of 9000. Put that in the record. The recurrent attacks were not of the type that we see so commonly in tuberculosis in the abdomen. We had a beautiful case a week ago today. The case was sent in as an appendicitis, but before we opened the abdomen we said it was not appendicitis, because the clinical history did not fit. We said that it was probably tuberculosis or some other complication. This woman had tuberculosis. We palpated a tuberculous mass running clear across the abdomen. Furthermore, it was not a tuberculosis of her omentum, but a tuberculosis of her mesocolon. There was a tuberculous ulcer in her colon. That is one of the reasons why a tuberculin test should have been made in this case. In that case we made the test and had a negative response. This is the third case of tuberculosis in the intestine that gave a negative response to the tuberculin test with a pronounced tuberculosis present.

We will make the usual incision for removing the appendix. We can lengthen it later if necessary. I insert my finger, and endeavor to pick up the caput coli. There is no Lane kink. There is the appendix. It is not the seat of an acute inflammatory condition. It is curled in at the under portion and knotted on itself, but it does not show evidence of an infection which would cause this elevation of temperature. We will remove it and have it examined by the microscopist to see whether it is a case of retention with absorption sufficient to give that leukocytosis. We do not believe it is. It does not look so.

There is a secondary mesentery present. That is an interesting manifestation. We have had a number of them recently. There is the mesentery; I pass my needle through that entirely and clear across so as to take up the peritoneum on both sides and then come back and catch the peritoneum on both sides, so that when I pull that suture taut I will have what? A complete circle of peritoneum. You will remember Wölffler found that was his weak point in making his gastro-enterostomies. I am getting this additional mesentery out of the field. Now we are ready for the amputation of the appendix.

One of the things I want to call your attention to in this case is the pathology on the surface of that caput coli. You see that vascularity all over the surface of the caput coli. That is pathology. I have not found the etiology of that condition, but the etiology is there because that vascularity is a sequence. It never occurs without a cause. We will put a ligature on the appendix. There can be no bleeding into the bowel from that. First, get a ligature around the appendix; second, a puckering string in the caput coli which to all intents and purposes is a Lembert stitch, and, third, a final Lembert stitch. This is the way I would close a bullet wound, and I am as careful and cautious in putting in this suture here as in closing a bullet wound. In following that plan we have had good fortune. We never operate in a case of tuberculosis that involves the base of the appendix. We never take it out.

Let me see why we have this vascularization. It is not from any lesion of the appendix. There is no evidence of tuberculosis in the ileum. No evidence of any lesion there. The vascularization shows that we have some type of inflammation in the neighborhood, either within or without the bowel. Let us examine this caput coli and see if there is tuberculosis present there. Here are some fixations extending upward; before I touch that I am going to return this intestine into the abdominal cavity and see if there is any trouble in the pelvis. We will examine, first of all, the most common site of trouble in the female, namely, the tube. The tube is not distended. It is a little congested, but there is no evidence of tuberculosis. There is some fluid in the pelvis. There is an infection, a submucous infection, somewhere. There is some thickening of the tube, but not enough for tuberculosis. This is not a tuberculosis of the tube. That is out of consideration. We will see if there is tuberculosis of the sigmoid. The transverse colon is away down in the pelvis. It is not the sigmoid. I am going to try and bring the sigmoid into the field. I can feel the uterus and the bladder and the tube on the opposite side, and a small cyst of Morgagni. That is of no pathologic significance. The other tube is not involved.

Now let us see why she had that recurrent pain in the upper abdomen and see if we can detect fixation or adhesions. Now you see how nicely we get on with this incision without cutting the muscles. There is some fixation due to adhesions at the hepatic flexure between the colon and the parietal peritoneum.

That is the cause of the repeatedly recurring retention. The gall-bladder can readily be brought into the field. It is normal. The pyloric zone of the stomach and the duodenum are also normal. The pylorus is not distended, nor is it thickened. It is perfectly pliable and soft. There is no white line or ulcer on the anterior surface. It is perfectly clear.

Here is the etiologic factor. These colonic adhesions come from the outer side of the abdominal wall. I do not know that we have accurately explained the etiology of these cases. We have been having considerable discussion about it lately, but to me it is not clear whether there has been an excessive irritation here. I will divide the peritoneum so as to get that piece out and let that bowel go down.

What about the constipation phase of this case, doctor? it has passed out of my mind.

The Intern: Her bowels were regular, doctor.

Dr. Murphy: There is the caput coli. See where this bowel was suspended! We put the carbolated gauze on these surfaces so that they will not re-attach.

Let me say again, with emphasis, that when you find that the physical findings and the analysis do not fit there is something wrong and you must make a more careful investigation. It is easy to make a diagnosis of recurrent appendicitis. That is a very common error.

Let the record show that on opening the abdomen the caput coli was easily drawn up, and that on its peritoneal surface it showed an area of vascular distention and connective-tissue development. The transverse colon was found in the pelvis. There was no tuberculosis there, and while there was some slight involvement of the tip of the appendix, it was not enough to make it pathologic. The right tube and ovary were then brought up into the field and inspected. There was no tuberculosis of the tube, nor was there congestion or enlargement of the vessels around the tube. The left tube and ovary and sigmoid were then brought over and showed no evidence of disease. The uterus was not surrounded by adhesions and was easily palpated. There was a small Morgagni cyst in the left mesovarium. It was found that the trouble in this case was due to fixation and adhesions at a level with the lower margin of the liver coming from the outer parietal peritoneum and suspending the colon and caput coli. These adhesions extended down to the caput coli like a network, and hung over and suspended it.

Let the record show further that the gall-bladder was examined and there were no stones; that the duodenum showed no evidence of disease, and there was no evidence of a gastric ulcer.

We have done all that is necessary in this case, and will proceed with the closure of the abdomen. The peritoneum is closed with a continuous catgut suture, everting the edges, making an ectropion, so that there will be no raw surface in the peritoneal cavity. That will prevent the formation of adhesions. Next we insert figure-of-8 stitches of silkworm-gut, completely obliterating all dead spaces.

The suggestion of Dr. Gerster, of New York, was very timely when, in connection with this class of cases, we were discussing the reason for failure in results in operations on the gall-bladder. One of the reasons to which he attributed poor results was that the incision was too small, and in our efforts for getting beautiful cosmetic results around the appendix we have in these chronic cases made our incisions too small and by doing so hazarded the patient's life. In the other cases in this class we hazarded the finding of the true etiology of the condition. We need larger incisions and more careful exploration after the abdomen is opened, and we need space in which to do that.

The skin-edges are approximated with horsehair, making a continuous suture. The wound is then dusted with bismuth subiodid, a desiccating powder, not an antiseptic, and sealed with a collodion gauze dressing. [This patient made an uneventful recovery, and at the time of leaving the hospital was free from all symptoms.—Ed.]

EXOPHTHALMIC GOITER

HISTORY

Female, single, twenty-one years of age, entered Mercy Hospital complaining of an enlargement at the site of the thyroid, palpitation of the heart, and nervous exhaustion. Four years ago she had typhoid, but otherwise has been free from serious illnesses until present trouble began.

In the spring of 1910 she noticed a slight enlargement just beneath the middle of the right sternocleidomastoid muscle. It increased steadily in size, and six months later she noticed a swelling in the midline and also somewhat on the left side of the neck. During the latter half of 1910 she worked hard. She felt that her strength was less than usual, and she became tired and nervous readily. When she danced or otherwise exercised she noticed that her heart would beat fast and forcibly, much more so than the amount of exertion would justify. In January, 1911, she was excitable and nervous. During the latter part of that month she began to have very severe headaches and felt exhausted. At this time she first noticed that the eyes were more prominent than usual. This sign increased in degree.

During February, March, and April of 1911 she was forced to stay in bed because of nervous prostration. At this time she often felt her heart beat forcibly, and she also had difficulty in breathing. She had intense headaches and very severe and prolonged attacks of vomiting, lasting often three to five days. She became emaciated, and could keep nothing on her stomach. She was placed on thyroid medication and kept quiet in bed. In June, 1911, she was able to be up and about, but suffered from nervousness and palpitation of the heart on the least excitement. Her condition has been about the same since then. It seems to her that the enlargement in the neck varies in size at times. Its growth, however, has been fairly constant, and it seems to be becoming of a firmer consistence. It never has been hard, nodular, or fixed to the surrounding tissues. Pulse ranges from 98 to 124. Urine is normal. Blood findings: Erythrocytes, 4,200,000; hemoglobin, 80 per cent.; leukocytes, 9,800; differential count: Polymorphonuclears, 52 per cent.; small mononuclears, 41 per cent.; large mononuclears, 3 per cent.; transitionals, 4 per cent. No eosinophils were found in a count of 200 cells. Blood pressure, 85 mm. of mercury.

COMMENTS AND OPERATION

Dr. Murphy: This is a fairly classic history of this lesion. There is a very definite relation between the clinical history and the pathologic findings in the thyroid. You can divide lesions of the thyroid into three grand classes: first, the lesion of the epithelial or secreting cellular elements of the gland; second, the lesion involving principally the blood-vessels; and, third, the lesion involving the connective tissue. So that there are three classes of lesions—lesions of the connective tissue, the lesions of metastatic infections with a hyperplasia of the connective tissue in the gland, with also some increase in the epithelial elements, pain and swelling, coming on usually as metastases from some other infectious disease, such as influenza, typhoid, and scarlet fever. This acute swelling continues usually for some weeks, then gradually subsides. If the inflammatory process is long in duration and severe, the connective tissue commences to contract. It can entirely obliterate the secreting cells in the gland and cause hypothyroidism.

I had two very striking cases of that class, one following tonsillitis, with an acute infection of the thyroid, edema, swelling, and sensitiveness. This patient I treated years ago, when she was a girl, for an enlarged thyroid. Later, after she was married, she developed some of the mild symptoms of Graves' disease, but under the arsenic treatment they disappeared entirely. About two years after that she was confined, and a year after the confinement she had a sudden severe tonsillitis which caused swelling and soreness in the lower part of her neck. She had an intense toxic nephritis, with blood in the urine, uremia with convulsions, unconsciousness, and blindness. From this she was relieved by an enormous blood-letting. This went off into a subacute condition, and about a year after the onset the edema and swelling subsided so that the woman fell off in weight from 215 or 230 pounds to 165 pounds. She had put on a great deal of adipose tissue within the three years preceding this metastatic infection; then, as her albumin diminished (but never disappeared), she had a second attack of edema and swelling of her limbs, and when I saw her she looked like a mass of blubber, filling a three-quarter-size bed and weighing over 300 pounds. The case puzzled me. Microscopic findings in the urine did not justify the conclusion that that colossal edema of her legs, arms, and abdomen was from her nephritis. She had a weekly or ten-day explosion of high temperature, and then it subsided again until she got another attack. That condition continued to recur. She had pain in the abdomen, with a profuse diarrhea. I saw her in consultation during my spring vacation, and it was two weeks before I arrived at a diagnosis, watching her every day and looking her over. Suddenly it dawned on me—what had become of her thyroid that had always been enlarged? Notwithstanding the swelling in her neck, I could determine that there was no thyroid. Then I said, this is a myxedema. Her eyebrows had almost disappeared, her hair, which had formerly been heavy, was just a wisp; her axillary hair was gone; her pubic hair was gone. This falling out of the hair was the key to the secret; it is a part of the general symptoms that go with myxedema. So I made a clean-cut diagnosis of myxedema. I put her on thyroid extract and she immediately began to improve and went down from 300 pounds to 120 pounds. She still had some albumin and some casts in her urine, but she got perfectly well. She again took up her social interests; she went to California the following winter and participated in everything that was going on, early and late. Suddenly she developed an acute septic endocarditis, then a pericarditis, and died in about three weeks.

Another patient was sent to me from Nebraska a number of years ago with much the same type of attack. I made a diagnosis of acute myxedema about six weeks after the subsidence of the swelling of her thyroid.

These are the acute manifestations, with the subsequent history of the lesions of the connective tissue.

There is another lesion of the connective tissue of the thyroid and that is the sarcomatous disease. Not infrequently sarcoma originates in the connective tissue of the thyroid.

We know that we can produce all the symptoms of Graves' disease, excepting the exophthalmos, by the administration of sheep's thyroid. They get the typical tremor and palpitation of the heart—all the symptoms of hyperthyroidism.

This hyperdevelopment of the vascular system is the principal lesion that occurs in connection with the vessels of the thyroid.

The important lesion—the common lesion—is a lesion of the secreting structures or epithelial elements and ducts of the thyroid. These lesions are divided into, first, the simple hyperplasia of the gland, an increase in the acini and an increase in the number of cells lining the acini. Second, a circumscribed increase, with an encapsulation, causing what is known as an adenoma of the thyroid. This is identical with the first lesion except that it is circumscribed and encapsulated. Third, multiple adenoma. Fourth, an increase in the secretion of the cells without absorption; in other words, an increase in the colloid material. When you cut into the gland you find a large mass of colloid material. That is the colloid goiter, which can be divided into the diffuse and circumscribed, and finally there is the combination of these two conditions.

We will operate on this patient now. We will make the so-called collar incision, extending across the neck from one sternocleidomastoid muscle to the other, and passing over the center of the enlarged thyroid. A crease in the skin is selected, so that the resulting scar will be less conspicuous. The incision extends through the skin, fascia, and platysma. The important thing in all these cases is to get, first, a good exposure, and, second, to have enough hemostats and have them on at the right time. The sternohyoid muscles are now exposed and must be divided so as to give us better access to the gland. I pinch the muscles with a hemostat before dividing them so as to minimize the bleeding. We divide these muscles high up close to the margin of the sternocleidomastoid, which we will also divide for a short distance. Now we have a splendid exposure of the thyroid.

Let the record show that this girl was very hoarse before the operation. I want that made a part of the record, because for some reason her recurrent laryngeal nerve was interfered with and we could not determine just what that reason was.

Now I commence to wipe off the tissues on the inferior margin of this gland; and now I come close on to the zone in which the recurrent laryngeal nerve is situated. Here is the inferior thyroid. The recurrent laryngeal nerve is in close proximity to that. There is a parathyroid! See that little yellow spot right there! Now the lower pole is fairly well loosened and we turn our attention to the upper pole. It is high up. We provided for that by extending the incision on that side away above the average position.

The recurrent laryngeal nerve is visible. There is no traction on the trachea. We are keeping close to the capsule of the gland all the time. That is the principal and only precaution that we can take to protect that recurrent laryngeal nerve. We are going to remove all of one lobe and a portion of the other lobe. I now incise the capsule of the gland. Now we will have use for many hemostats because we wish to have as little hemorrhage as possible. As fast as we grasp a portion of the gland with a hemostat we excise it. After we have removed the desired amount of gland tissue we will ligate every bit of the stump in the grasp of these hemostats with catgut. Before suturing the capsule over the stump we will search carefully for bleeding points and ligate any that we may find. Having done so, we are now ready to suture the cut ends of the muscles together, using catgut.

I am sewing up the sternohyoid muscles just where I divided them, and also the nick in the sternocleidomastoid. Deep catgut and superficial horsehair sutures approximate the wound edges without tension. A gutta-percha tissue drain is passed down into the lateral angles of the wound and emerges in the midline.

The other day I made a ligation under a local analgesia. That night the boy had a temperature of 103° F., notwithstanding the small incision, and a pulse of 148. I merely ligated the upper pole. His has been a desperate case. We had him here for a number of weeks getting him ready for an operation. He had a little diarrhea; his pulse went to 160, and he almost died. These patients do not stand anything; you are afraid to touch this class of cases. You get them on the borderline, just ready to die. We have them come in from the country in the early fall, never touch the patient, and he dies in a week. We keep them still, apply the ice-bag, and do everything else necessary, and still they die, showing how desperate these cases are. This is the class of cases for which there is no control; if you give them adrenalin they get intensely intoxicated and they may die. Give them a little morphin if very restless—that helps them.

Now we have the normal conformation of the neck restored. The wound is sealed with gauze saturated with collodion, and a large dressing is applied to absorb the wound secretions.

Now let us go back to the pathology of this case.

I was speaking of the excessive secretion in the cells and acini. Where this is still more circumscribed and where you have much secretion without absorption in the circumscribed area, you may have, as a sequence, a circumscribed cyst. This cyst first contains colloid material, or the normal secretion from the cells of that particular acinus. As the quantity of secretion increases it produces a pressure on the cells, causing them to flatten. With change of conformation of the cells comes a change in the character of the secretion. The secretion rapidly ceases to be normal colloid material, but becomes serous material. When these cysts are opened they are found to be monolocular, rarely multilocular. You may have two cavities contacting with each other, but not communicating. That is in conformation with the law of change of secretion in every epithelial secreting gland. As the change in the cell conformation takes from the cell all its normal physiologic secretion, it gives it the character of a serous secretion in place of an epithelial or glandular secretion. Occasionally there is a hemorrhage into this cyst and there develops what is known as a grumous cyst of the thyroid. These are only changes in the product and not changes in the tissues themselves at all. The most common position is in the isthmus, with its tip in the gland.

I remember a doctor who had one of these cysts, and he almost suffocated before he got to the hospital, where the late Dr. Alexander Hugh Ferguson was operating. Dr. Ferguson opened the cyst, that was all that was necessary to do, and drained it. It contained grumous material. The process of repair went on, and cicatrization occurred. That was a cure as well as a relief of the danger to the man's life.

The type we have under consideration now is the increase in the colloid material, and an increase, as we believe, only of a physiologic product, having in this particular individual a very deleterious effect on the central nervous system.

The Graves' disease syndrome of symptoms is always recognized. There is one time, however, to recognize it, and that is long before the exophthalmos appears, and long before the gland is large enough to be seen by everybody in the block except the doctor, and long before the patient has a grave intoxication of the central nervous system, and long before the heart is impaired so seriously that it is a very dangerous thing to do anything with the patient. It is not necessary for us at this day to watch for all these grave manifestations before we recognize and before we treat the disease. I think it can safely be said that Graves' disease, from its very incipiency, is a surgical disease, and that our hope in the future in Graves' disease is going to depend on timely operation, and we are going to be responsible for acting in time. The results in the treatment of Graves' disease through surgical management are illustrated in the work of Charles Mayo and Theodore Kocher, and of many others who have operated on the thyroid with splendid success. So that there is no longer any question as to the result of surgical treatment. We know, from a clinical experience running over a quarter of a century or more, that there is no question as to what the previous results were with medical treatment, so that the surgical treatment is the only treatment that is indicated in this disease, but it must be timely. I think this is one of the diseases where the surgeon should not touch the late case. When they get to that serious condition, they must not be operated. Put them in bed, elevate the foot of the bed, put on the ice-bag, and give them sodium cacodylate. Keep them absolutely still. That does not mean get up half a day or have company half a day, or read correspondence, or get up and go to the toilet—nothing of the sort. Just as in the case of this boy we have here now, carry them along and you get to the stage where you can do one procedure after another.

I recall a patient who came to see me in what I consider was the early stage of the disease—about six weeks after the initial manifestations. The girl had learned of a serious breach of family obligation on the part of her father, and it depressed her nervous system so that in two weeks she had an enormous pulsating goiter, heart-beat 160 to 180. To touch that girl at that stage would have been fatal. We put her in bed, and put an ice-bag on. The family doctor who had charge of her says he carried out the instructions to the letter. In fourteen months that gland was contracted, heart-beat down to 80, and she is back again to her normal condition, with a fairly firm and somewhat enlarged thyroid gland. The firmness of that gland is due to the cicatricial contraction around the acini, which were enlarged.

A sudden contraction after a gland has been large is a serious matter. I can illustrate the seriousness of that situation by citing a case. The patient came over from Michigan with an enormously enlarged gland and in quite a serious condition. We kept her for three months on the ice-bag and rest treatment. She improved so that we could perform a ligation of both the upper poles. She improved rather rapidly and went home. That was in February. When I returned in August I had a letter from her saying she was not well, and that she was coming to Chicago. The gland was found to be still large, and just as hard as a piece of iron. This started up a thrombosis in the vessels in the gland. Then it began to contract, which made it so firm and hard, obliterating all the secretion from that gland, destroying it as a secreting gland, and she developed a myxedema and died within a week after her arrival. That shows you one of the dangers of contraction of that connective tissue, or thrombosis with subsequent contraction and obliteration.

In this variety of goiter, therefore, you have to take into consideration when you look at the gland, first, which of the three tissue elements are involved; second, if they are involved, are you having the more serious stages of Graves' disease, and is there produced more thyroid secretion than that system can care for?

In the treatment of hyperthyroidism many questions have come up as to the medical treatment. I have seen 100 or more of these patients get perfectly well without surgical intervention, and I have seen a number of patients with Graves' disease get well without surgical intervention. With rest, ice-bag, arsenic, and no mental irritation they practically all get well if they are of the typical hyperplastic type. That class of patient gets well on medical treatment. That is a class of cases that is absolutely medical in the beginning. There are patients who get tremor when they straighten out the fingers, which is one of the very early signs; they get tachycardia long before they get exophthalmos. If they get that, operate then, before the heart muscle cells are thick. Operation in the early stages does not cause a mortality of 0.5 per cent. It is one of the safest operations in surgery. Operate in the late stage of Graves' disease, and it is one of the most dangerous operations. You are absolutely at the mercy of the patient and the intoxication.

TRAUMATIC LESION OF BRAIN

HISTORY

The previous history of this patient is of no bearing on the lesion for which he now comes to us.

On March 14, 1912, about two months ago, he was struck by a fellow laborer with a tamping pick over the right postparietal region accidentally. He fell to the ground and was unconscious for a minute (?). When he recovered consciousness, he was weak and nauseated, but did not vomit. He bled profusely from the scalp wound, but did not bleed from ears, nose, or mouth. The doctor who was called sutured and dressed the scalp wound, but did not explore the skull beneath for evidence of depression or fracture. There was primary union in the scalp wound.

The patient was not put to bed, but has been up and about every day since the injury. About a week after the injury he developed occipital headaches of a throbbing character. The pains radiated from the right occipital region down the neck. When the headaches get very severe, the patient is unable to hold his head erect. No motor or sensory disturbances, except a numbness in the left index-finger. This, however, is not continuous. No double vision; no loss of memory; patient answers questions intelligently; some vertigo and there is a sense of pressure over injured area; reflexes normal.

COMMENT AND OPERATION

This is a typical history, with a few exceptions. The principal exception is the speed of onset of the symptoms. In traumas of the skull, even with fractures that are demonstrable, the rule is for the patient not to have the headaches, the vertigo, or the sensory or motor disturbances so soon after the injury unless they occur instantly from a trauma of the cortical portion. They usually come on many months and sometimes years after the accident, and the later they are in coming on, the more grave and serious they are.

Management of skull injuries—of the concussions that render patients unconscious, of the contusions with depression of the skull, and of the depressed fractures of the skull, and of fractures of the skull at the base—is one of the dark chapters in surgery,—dark so far as the *ultimate* outcome is concerned,—and one of the bright chapters in surgery so far as the *immediate* effect is concerned. That is, patients may have very severe injuries,—compound fractures and comminuted fractures, with the escape of large quantities of brain tissue, with the skull fragments driven down into the brain for an inch and a half (which are removed),—and the patient may recover immediately from the accident and remain well for a long period of time.

The injuries with compound or depressed fractures are the least injurious; that is, where the edge of a sharp instrument penetrates the brain, like the claw of a hammer, a nail, or scissors, if the patients recover from the immediate effect—and they do with proper treatment—you rarely have any trouble subsequently. But the blows which are sufficient to produce unconsciousness, with or without a fracture, never leave the patient free of the danger of a cerebral complication or a nerve complication for all time. The period of time that may elapse before the serious symptoms come on varies greatly. The serious symptoms sometimes come on within a few weeks, or a few months, and again not for many years. I remember one man who had his first convulsion twenty-seven years after he was kicked on the head by a horse. He had a depressed fracture, compound, which was elevated at the time and healed. Many years ago I had a patient who fell from a second-story window. The child was unconscious for three weeks; it had a comminuted fracture of the skull. The child got well, developed into a man, advanced to a very prominent position in a bank, and twenty-two years after the accident he had his first convulsion. Then he had convulsions every few weeks, and he is now completely incapacitated by his convulsions.

I cite these cases in order to impress on you the teaching of the late Dr. Fenger concerning the management of skull injuries, which should be a more general practice with the medical profession. Fenger realized, I think more than any other man, the significance of a blow of any kind on the head that rendered the patient unconscious, with or without a fracture. It was Fenger's teaching, when I was an intern in the Cook County Hospital, that every man who came to the County Hospital who reported that he had been injured sufficiently to render him unconscious should be put to bed with an ice-bag on his head and kept in bed six weeks, regardless of what his symptoms were, so as to give his cerebral tissue and his meninges an opportunity to return to the normal with the least deposit of connective tissue. Fenger impressed on us the idea that these cases many years later would manifest symptoms, and that if we hoped to avoid these symptoms, they were not to be avoided so much by the operation as by the quiet treatment which these patients were to have immediately after the injury.

We have no idea as to whether this pick went through this man's skull and whether it injured the meninges as a direct result of the trauma. We do not know. We do know, however, that this man has pronounced signs of local irritation, either from the trauma, from the direct penetration, or from the concussion, and we also know that these headaches, once they start, usually lead to very serious manifestations. The most pronounced symptoms are, first, the recurrent headaches; after the recurrent headaches have continued for months or years come the attacks or convulsions, and then the grave attacks of epilepsy, and once the patient has that type of epilepsy, he is practically doomed. In a large experience we have had only one case in twenty years where a cure followed operation in a grave case of epilepsy. We have had some very strikingly good results, and we have had a great many very lamentable failures, but in talking to these patients when the epilepsy has come on I say to them plainly, "Your chance for recovery by doing an operation on your brain is not very promising."

The type of operation varies. Originally we merely took out the irritating spicule of bone. We just took out the depressed fragments of bone, and if we did not find them, we would be discouraged. In later years we learned that removing the depressed fragment of bone has little or no effect on the symptomatology in these cases. We now take out an area of dura that has been traumatized and that has given us the most pronounced relief.

I recall an incident that occurred when I was a student in Rush Medical College. A French physiologist came to America making certain demonstrations regarding irritations of the dura, and one was that in a pigeon he could produce epileptic convulsions by the slightest electric irritation of the dura, and it has always been my belief that it is the dura that is the irritating zone. It is my belief that the connective-tissue deposit in the dura produces a type of irritation that causes the convulsions. That is why, in cases of fractures of the base with convulsions, we never do an operation because we cannot remove the dura in that zone.

[To Assistant.]—Where has this patient been marked? We do not want to take out the wrong side, an accident which is very frequent. Remember, the wrong eye has been taken out, the wrong leg has been amputated, the wrong Gasserian ganglion has been removed, and the wrong side has been operated on for hernia because of a lack of system in marking. *If you have a system, that is what counts and saves trouble.*

The head has been shaved and painted with tincture of iodin. A rubber constrictor has been placed around the head just above the ears. We wish to locate, first, the Rolandic fissure. Take half the distance between the occipital protuberance and the base of the nasal process of the frontal bone, add half an inch backward. That is the line. Just back of the Rolandic zone.

That zone will form the center of our flap, which will be nearly circular, base down, measuring about two inches at the base and four inches in its widest diameter. The only evidence of the injury is a very slight scar, barely visible. His veins are dilated considerably, and that is where the bleeding we see comes from. The slightest pressure on the skull stops it.

I recall a case that came to me from Oshkosh, Wis. The patient had been injured eleven years before, as I remember it now. He was sleigh-riding, the horse ran away, the cutter tipped over, and his head collided with a telegraph pole. He was unconscious for four or five days. Then he apparently got well and went along nicely. He went to school; had the usual work. Some years afterward he began having intermittent headaches. They became more and more severe. Finally they became so severe that he had to be taken out of school.

We first operated on him by removing a small depression that existed, with a cyst under the depression. He improved and remained fairly well for I forget how many months, when he commenced to have headaches again. One day he had three convulsions, became unconscious, and remained unconscious. My associate at that time, Dr. Lemke, went to his home and saw him. He drilled through his skull, tapped his lateral ventricles, which relieved the internal pressure, and he was better for the time being. He remained well for a few weeks, when he had another convulsion. They brought him to Chicago, and I operated on him for the third time. In the second operation we removed a large area of the dura. We made an incision something on the type I am making now, and removed a large area of the dura. He recovered, and when I heard of him some years afterward he had neither a return of the headaches nor of the convulsions. That is a striking contrast between the drainage of the ventricles on the one side, the removal of the spicule of bone on the other, and the removal of the dura, on the third.

Since that time I have made more extensive removals of the dura than we ever did before, and since that time I can say that my results have been improving, but they have never been gratifying—they have never given results like those obtained in other phases of surgery. Of course, you must admit in the beginning that no one knows yet what the true etiologic factor of epilepsy is, and our chances of bettering the situation very much are not great until we get a more precise knowledge of what epilepsy is. We know some of the conditions that produce it—that is, a few.

Having turned down this flap of muscle fascia and skin, we will make a trephine opening and then chip through the skull to make the bone-flap. This is a very thick skull in this neighborhood. For everyday work the DeVilbiss instrument is the best; for extensive work, the Neff instrument is the best.

We have completely outlined the bone-flap and shall turn it down by fracturing it at its base, without detaching or removing its blood supply. We always make an osteoplastic flap. I do not have to cut the scalp. I leave it intact. I crease the bone in the direction in which I wish to have it fracture. One must be careful not to penetrate the dura. Now I will free the adherent dura from the bone-flap.

Let the records show that the dura bleeds profusely everywhere; in place of the ordinary free white membrane one sees when the dura is normal, see how it bleeds, showing the inflammatory condition that has been present here. Probably as the result of the trauma the dura was separated from the bone and has been developing new vessels for the purpose of reattaching itself to the bone. You see the pick did not come through the skull. There is no evidence of any injury of the inner table of the skull where he was struck, showing that the effect produced on the inside was a concussion effect and not a penetrating effect.

You will remember the splendid paper by Dudley Allen, of Cleveland, written some years ago, in which he analyzed a large number of cases of skull injury without penetration of the dura and most of the patients eventually found their way into an asylum. Many of them had subdural irritation.

I want to take out a certain zone of that dura, but I must leave enough dura not to overlap the margin of that bone-flap when it comes back, otherwise the flap will sink down. Note the thickness of that dura. It is at least three times as thick as the normal dura. Of course, that is in consonance with this finding on the outside. The pia is also thickened. The cortex beneath is edematous right under where the pick struck. The edematous area is very evident and is highly edematous.

That is one of the prettiest things I ever saw in all my head surgery. Beyond the zone of reaction the dura is normal. There is an inflammatory condition around the vascular network, with an exudate. He evidently had a hematoma beneath and an inflammatory condition. There was no evidence of a depression, nothing when I lifted the skull, except this extreme vascularity, which attracted my attention. Formerly, if we found no depression we let the dura alone.

I will excise the thickened portion, thus removing the focus of irritation. We will not go beyond the line of the osteoplastic flap, because if we did, the dura would again become adherent. We will leave a rim of dura all the way around on which the flap can rest and not sink in. I want to put in a piece of paraffin to prevent adhesions from forming. A thin piece of paraffin will answer the purpose. Now I have the dura free. That piece of paraffin will stay in there and it will become fixed. It covers all the brain surface which we denuded of its dura.

Now we can see the reason for all this man's symptoms, even in the gross pathology demonstrable.

Visiting Surgeon: What is the length of time before that paraffin will disappear?

Dr. Murphy: It will never disappear. This is a coal-tar paraffin. If it were a whale-oil paraffin, it would become absorbed. Mineral paraffin is not absorbable at all. We have often had to remove it when it was used for correcting facial deformities. Then, again, we have had to remove it many times years afterward when operating for the cure of a hernia where paraffin had been injected into the abdominal wall over the internal ring. There was no effort at absorption; the paraffin became encapsulated. And the capsule around it looks like a serous membrane—smooth and glistening. That has been one of the things that led me to consider a plan which I am going to put into execution when I get to the first joint where I cannot get sufficient fat and fascia to cover the articular ends of the bone. I am going to cover the raw end of the bone with paraffin—just a very thin plate of paraffin—and let it stay. If the paraffin were strong enough to carry the weight, we would put it between the bone-ends to prevent union. It is never absorbed; the granulation tissues do not grow into it, and it would insure earlier mobility and it would be very much less painful than the plan we follow now. It is like the transplantation of fascia flaps taken from a point distant from the joint. We never do it until we are forced to do so, because we have been getting grave results. We had one case of knee-joint ankylosis in which we took the interposing flap from the fascia lata over the trochanter major. We took enough to cover all the bone surface, and did not have to use the paraffin. When the time comes that we cannot do it in that way, then the paraffin is in readiness. Dr. Taylor, of Baltimore, has recently been using wax; Baer used hog's bladder, and that is, of course, eventually absorbed, but the idea is to use a material that is not absorbed.

Let the records show that the pick did not penetrate the skull; an area of parietal zone showed great vascularity just beneath the point where the pick struck the skull. There was firm fixation of the pia and dura and a cystic formation over an area the size of a half-dollar. These adhesions were soft and separated easily, and the pia and dura were freed entirely to the midline. A moon-shaped portion of the dura was taken out, and a moon-shaped piece of paraffin was inserted between the dura and pia extending clear to the septum. This was secured in position with catgut passing through the dura. A portion of the moon-shaped flap was removed over the paraffin plate so that the paraffin will come directly in contact with the bone. Then the osteoplastic flap was put back in position and fitted accurately into its place. Through this means we hope to prevent the pia from becoming adherent either to the bone or to the dura at the point of irritation. There is no encapsulated cyst beneath. The soft tissue flap will be sutured with horsehair. The head will be encased in a starch bandage, and the patient will not be permitted to move for five weeks. The case will be treated as a trauma of the skull.

TRIFACIAL NEURALGIA

HISTORY

Housewife, aged forty-four. Entered hospital because of severe pain in right side of head and face. Family history and previous personal history negative.

In 1892, twenty years ago, patient says her teeth were badly decayed. By the time she was thirty-seven years old more than half of them had been removed. None had been ulcerated. She does not think teeth of right side were more affected than those on left. She had suffered from toothache occasionally, but never to an excessive degree.

In September, 1905, seven years ago, she was seized suddenly with a lightning-like flash of pain on the right side of the face. It was so severe that she screamed. The pain disappeared just as suddenly as it came. In a few minutes another attack of pain came, but it disappeared just as did the first one. It left no soreness. Another attack of pain came in two days, the next in ten days. During the next two years the pains would come at intervals of days or months, and during the two years following this she was free from pain of any kind. At the end of this period, four years after the first attack, the pain recurred, but the character of the pain changed. Often it would come on gradually, getting worse and worse, until it became unbearable, and the patient would drop to the floor, writhing and screaming in agony. After one or two minutes the attack would pass off and she would resume her work, but feeling exhausted and unstrung.

Eating or talking is especially apt to precipitate attacks. The patient moves lips and jaws very slightly while talking, as though the jaw were ankylosed.

Two and one-half years ago she entered this hospital and was injected on the right side by Dr. Murphy. The patient states that alcohol was used. For nine months freedom from attacks was absolute, but soon the pains returned and were as bad as ever. She suffered for two weeks and then entered another hospital in this city, where a second nerve injection was made. It had no effect. During the last year and a half she has never been free from attacks of pain for more than a week or a month at a time. The face is reddened on the affected side only during the attack. The right side of the face is hyperesthetic about the middle of cheek, just below the eye, and on the chin it is analgesic. She never has contracted a drug habit. Never has had any trouble with the nasal sinuses, eyes, ears, or throat, nor has she been injured about the head or face in any way. No muscle paralysis or facial distortions are apparent. Examination of heart revealed nothing pathologic. Urine contained a trace of albumin.

Examination by Dr. Murphy: A strip one-half inch in width, extending from angle of mouth upward and backward, is sensitive and causes spasms of facial muscles when irritated by a pinprick. The region supplied by the infra-orbital nerve has not regained its sensation. The region supplied by the submental nerve is devoid of sensation.

COMMENTS AND OPERATION

Let the record show that this is a classic history; that she has had the periods of relief mentioned from the alcohol injections; that the injections ceased to give her relief.

We will make an osteoplastic flap, consisting of scalp and bone, in the temporal region, with the base at the zygoma. We must be careful not to injure the middle meningeal artery. The dura will not be opened, but is very carefully stripped from the middle fossa of the skull until the inferior and superior maxillary divisions of the nerve are found. These are traced backward to the ganglion. A rubber tube constrictor incircles the head just above the ears. This effectively prevents hemorrhage from the scalp.

The danger of injury to the brain is, I believe, lessened by this operation. Here is the middle meningeal artery now.

We are right at the foramen ovale, and both nerve-trunks are plainly in view. We will cut these nerves and then put some beeswax—a thin plate—over the foramen, so that the stumps of the nerves cannot unite with the distal portion. There is considerable bleeding, so that we must proceed slowly. As I free the nerve I elevate it with a nerve-hook and then sever it with a knife. In that way I am sure that all the fibers of the nerve have been divided. Beeswax is better than paraffin for this work. The wax goes down as a peg into each of these foramina, and then spreads out above the peg so as to cover the entire opening. The brain will come back as soon as I release the pressure on it. I like the Horsley instrument for this purpose. It is a great protection to the brain. It certainly lessens the chances of doing injury with the retractor.

We are now ready to replace the osteoplastic flap. Remember that the dura has not been cut. We simply allow the bone and fascia flap to fall back in place and suture the skin with silkworm-gut and horsehair, without drainage.

These cases can be treated in a variety of ways: First, we have the injection method; second, the division method with the injection; these are extracranial procedures; third, the intracranial types of operation, the division of the roots inside the foramina, and the closing of the foramina with heterogeneous material, such as the rubber tissue of Abbe, the screws of Mayo, and ligatures; the wax plug of Murphy. Next is the removal of the Gasserian ganglion and the removal of the sensory root of the ganglion—Frazier's method.

Osmic acid injection is the plan we usually resort to first, with the division of the nerve at the intracranial foramina. It gives a relief varying from fourteen to eighteen months. Patients who have gone as long as seven years without recurrence are considered cured. A number of them have gone three years, a smaller number four years, but the average case recurs in from fourteen to eighteen months. The second osmic acid injection gives about the same period of relief as the first. The third or fourth injections are not so successful. The alcohol injection gives relief for from six to ten months. Whether the injection is given above the zygomatic arch or below it seems to make but little difference. To us the injection below the zygomatic arch seems a safer and more certain procedure.

We have done altogether some 16 or 18 Gasserian ganglion removals, that is, complete removals, securing in all cases in which we have been able to get reports permanent relief. It is the desirable operation if it can be executed, but we have found it difficult to execute on account of the bleeding, and therefore we have resorted to the easier and safer procedure of extradural exposure of the inferior and superior maxillary division of the nerve. We have divided them and placed a paraffin or wax plug a little larger and thicker than a nickel over the foramen, and pressed it in and firmly interposed it in that position. The pivots which are made by pressure prevent the material from sliding and those pivots are in the foramen ovale and foramen rotundum. The superior maxillary division is not disturbed in this procedure.

In the removal of the Gasserian ganglion the roots are not divided until the upper layer of the dura is entirely freed from the ganglion; then the roots are divided and the Gasserian ganglion evulsed, holding both of the roots in the same root-retracting forceps, which is of the pattern of the Mayo cystic or common-duct forceps. It makes a splendid evulsor, for the depressed root is removed when it passes through the upper half of the dura on the surface of the ganglion and then backward and upward over the temporal elevation until the posterior root is seen shining in the field. This is hooked up with a blunt hook resembling that used by Alexander in extracting the round ligament, or by the oculists in doing myotomies on the eye muscles. When the root is thoroughly hooked, traction is made on it. It is not cut off, just simply dropped, because it has been detached from its central position; it never reunites because there is a permanent degeneration of the part, that is, the pons having no neurilemma, its axons are permanently incapacitated, as was shown in the experiments of Frazier.

A starch headcap will be put on in this case and not removed for two weeks, when healing will have taken place.

TUMOR OF SPINAL CORD

HISTORY

Female, aged fifty, housewife. Father died of arteriosclerosis at the age of seventy-four. Mother died from nephritis at the age of fifty-nine. No history of malignancy or tuberculosis in family. Menstruation began at fifteen, regular up to thirty-five. At the age of thirty-three patient began passing blood-clots with each menstrual flow. This lasted until she was thirty-five, at which time she had a miscarriage. Following this she was cureted, and since then she has not menstruated. Had one child at twenty-eight, normal labor and puerperium. No laceration.

Had measles, mumps, and whooping-cough when a child. Following her miscarriage she had a severe infection due to the curetage, and was in bed for three weeks. Three months following she had a severe pelvic infection which subsided in a month. There was no operation at this time. Two years following this patient had an attack of bronchitis for three weeks.

Present Trouble.—In February, 1908, she had intermittent attacks of pain in the right elbow and forearm. Patient thought it was due to rheumatism. The attacks came on especially after writing much; were more frequent at night, often causing the patient to awake from sleep. These attacks of pain occurred on an average of one every two or three weeks, without change in character. In December, 1909, patient began to notice a numbness in her right hand, involving the fourth and fifth fingers. This numbness gradually extended up the ulnar border of the arm.

In January, 1910, she was examined by a doctor and the sensation in the right arm was more pronounced on the ulnar border, extending to the shoulder, also in the right leg extending from the toes up to the waist-line, and in the left leg from the waist-line to the knee. Since this time the numbness has gradually increased until sensation is entirely absent from the toes to the waist-line, and there is partial anesthesia from the waist-line up to about the third rib, and in the arm and fingers to the shoulders; more pronounced on the ulnar side. The first disturbance was noticed December, 1909, when, on attempting to step into a carriage, she noticed a heaviness in the right leg. This gradually increased in degree until now she cannot use either leg. Since the patient began to notice the trouble she has not had any difficulty with bowels or bladder. There is no disturbance in eyes or ears. The patient was operated on at that time, but in February, 1912, the symptoms recurred.

COMMENTS AND OPERATION

Yesterday we operated on this patient for a tumor of the spinal cord which was situated between the spinous processes of the seventh and the sixth cervical vertebræ. It was a recurrent case. The patient originally came to Dr. Hugh T. Patrick, who examined her, and he was unable to make a diagnosis of tumor at that time. Some three months later a diagnosis of tumor was made. Then she went along for six or seven months before she was operated on. The symptoms she had originally were pain in the right and left arms, then pain in the limbs, followed by hemiplegia and then a paraplegia, so that she was practically paralyzed, except for a few muscles in her upper extremity. The tumor was situated between the sixth and seventh spinous processes of the cervical vertebræ. At the first operation it was situated between the fifth and sixth, shutting off the nerve-supply to the brachial plexus, except the two upper roots.

The trouble was diagnosed as a tumor on the posterior surface of the cord. We cut down on it at that time,—about two years ago,—and removed a tumor that came out in the posterior fissure of the cord. It affected sensation first and then motion. The tumor was about an inch and a half long, and in diameter about the size of my little finger. We lifted it out without traumatizing the cord, and the woman made a complete and perfect recovery. She resumed her usual work; in fact, she was well part of last winter, so that she did not have a maid and tended the furnace. This shows you how fully restored her function was. Then, about the middle of January, 1912, she began to have discomfort in her arm, the pain began to increase in severity, extended higher up, until finally she got ataxia of gait. Dr. Patrick concluded that she had another tumor, and that it was lower down than the first—situated between the sixth and seventh spinous processes of the cervical vertebræ and on the right side.

We operated on her yesterday, reopened the spinal cord, freed it from cicatricial tissue, and took out a second tumor, about half the size of the original one. We closed the cord membranes carefully with fine catgut sutures, then put in deep and superficial catgut sutures and coaptated the skin with horsehair. A collodion gauze dressing sealed the wound. It was situated near the posterior root of the spinal nerve and pressed on the anterior and anterolateral columns. After it began to grow the motor symptoms were more pronounced on the right side. The operation was done without any untoward symptoms. Last night she felt better than she had for months; she did not have any pressure pain in her cord. She will undoubtedly go on to recovery. The tumor proved to be of the endothelial type, originating from the lining of a lymph or blood-vessel. These tumors are really of the nature of sarcomata. Whether this tumor will recur or not we do not know, but I should say it probably will; but if recurrence does not take place in two years, or in a year and a half, we shall feel that we have been well rewarded for the effort. It is a better reward than one usually gets.

[When the patient left the hospital, about three weeks after the operation, the wound had healed per primam and she was apparently entirely well and free from pain.—Ed.]

CHRONIC MASTITIS

HISTORY

Housewife, thirty-two years old. Entered Mercy Hospital because of a tumor-mass in the left breast. She has been married fourteen years and has borne four children. She has had two miscarriages at about the third month each, following overwork. She always has nursed her children. Past life has been free from all sicknesses of a serious nature. A cousin died of carcinoma of the breast.

Ten years ago (1902), while nursing her second child, she had an excessive amount of milk in both breasts. She wanted to check this excessive secretion, and for this and no other reason, she is sure, she put the baby to the right breast always. She states that within four weeks the left breast became somewhat hard and slightly tender. This tenderness persisted and increased in severity, but she thought that the breast had "caked" because she did not put the baby to it, and therefore paid little attention to the tenderness. Four months after the baby was born the breast had become so sore that on the advice of a neighbor she applied a clay pipe to the nipple and sucked out several tablespoonfuls of a material that she feels sure was thick pus. It had a sour, milky odor. The breast (left) remained tender and firm beneath the nipple and to its outer edge. This has remained so ever since.

One and one-half years later she became pregnant for the third time. The breast became slightly more tender, but no milk flowed, nor did it discharge again. Her fourth child was born within two years, and the breast was the same as before, with the exception that the mass was a little harder and a little larger. At no time since its first appearance has the tenderness and firmness disappeared.

In the fall of 1909, while in a crowded street, she was struck a sharp blow on the breast by a person's elbow. For one-half hour she suffered considerable pain. She cannot remember that the breast was more tender than usual during the following day or two. She thinks that the mass has increased in size and firmness very gradually, but that it is less tender now than during the years when she was having children. The skin never was discolored or inflamed at any time. The tumor was never nodular or rough. The axillary glands cannot be palpated. Four months ago (December, 1911) she first noticed that the nipple seemed to be held firmly to the hard mass beneath it. The mass is now of about the size of a lemon, is very firm, and seems to be fixed in the breast substance as well as to the nipple and the skin about it. Heart, normal. Urine, negative.

COMMENTS AND OPERATION

DR. MURPHY: As I remember, there was a yellowish discharge from the nipple. That should be put in the history, if it is not there. You cannot interpret that as pus, because then you have a thick, yellowish discharge from the lacteal glands. It is not at all uncommon. This history is exceptional on account of the period of time that elapsed between the trauma and the manifestation of the tumor. It is three years since she had an injury. You will note again that she had only one injury, not many injuries; that following this one injury she had considerable pain for half an hour, then it subsided, and the whole thing disappeared. When was the discharge from the breast?

THE INTERN: Three years ago.

DR. MURPHY: Before the injury or after the injury?

THE INTERN: A year before the injury.

DR. MURPHY: She never had a suppurating breast. She nursed all her children preceding that time, but not the last one, and with the last child she had no mastitis and no inflammation. The breast was a little tender, but the pain was not severe. There was no suppuration. A year after that time she received a trauma, and two years after that time she comes here with a lesion in her breast.

The question arises, has this woman, as the result of the trauma, a carcinoma? Has she a sarcoma, or has she an interstitial mastitis? What is her temperature?

THE INTERN: The temperature is normal.

DR. MURPHY: There is evidence of acute infection. We examined this woman's breast yesterday and we found that she has a diffuse infiltration of the breast. When the nipple is retracted, the skin over it is fixed. She is only thirty-two years of age, stout, ruddy, and robust. That means what? That means, if we interpret this as a sarcoma or carcinoma, that we have to deal with a condition that is ultimately going to terminate in a cancer death. Then, were this woman sixty-two years of age, she would have a much better life expectancy than she would have at thirty-two. When a woman of thirty-two comes to you and is stout, with a lesion in her breast that has existed for a considerable period, practically in 100 per cent. of the cases she will have a cancer death. That is an admission, but it is a fact. When you find a tumor in such an individual, you base the life expectancy on the individual rather than on the statistics. Women who are stout, who have a large amount of fat, particularly when they are young, have rapid transmission of the malignant epithelial cells through the lymphatics. On the other hand, if they are thin and aged, the lymphatic circulation is slower; the new connective-tissue formation encapsulates and retains the epithelial cells *in loco* a much longer period of time. That is what one must assume in making a prognosis in such a case as this.

There is here the typically retracted nipple. There is a mass under it. It has a sense of fluctuation. There is no evidence of involvement of the axillary glands as yet. We are going to take out this tumor as it is, then bisect it, examine it, and determine what we will do with the axillary glands. This mass must come out, because whether it is malignant or not, there is only one treatment, and that is its removal and its complete removal at this time. We will have skin tissue in great abundance.

In making the skin incision in this case we are not going into the axilla, but above it. It will encircle the breast. The incision will extend down to the pectoral fascia. I will not take out the pectoral muscle at this time, but will remove the aponeurosis of the pectoralis major muscle. In doing these operations we take into consideration the line of lymphatics leading from the tumor and the position which the tumor occupies in the breast. The incision curves above the muscle, so as not to have the scar line crossing the axilla. The fascia between the pectoralis major and pectoralis minor should always be removed because it contains lymph-nodes which may be the seat of metastases now or become such later. The next thing is to take off this breast and make an examination of it before we attack the axilla. This is done by freeing the breast from the chest-wall, removing all the fascia containing lymph-vessels and lymph-nodes. We will now incise the mass. It contains a purulent fluid. It is not necessarily pus. It is questionable in my mind whether that tumor is malignant. This breast is riddled with infiltration and degeneration, and it contains either pus or it is a retention of the secretion from the epithelial cells lining the lactiferous glands and ducts. We will not take out the axillary lymph-nodes at this sitting. The multiple "infections" here are accumulations which are probably retention cysts with mixed infection. We will have this mass examined microscopically, and then, if necessary, if it shows signs of malignancy we will take out the axillary glands later.

This woman is very stout, and the removal of fat from her axilla and the removal of the lymphatics of the axilla will give much greater edema and swelling of the arm even with the implantation of the pectoralis major than would occur in the average case. That is why we do not wish to go into the axilla unless this tumor of the breast is malignant. If this woman should get lymph stasis, stout as she is, her arm will become practically useless. We have not disturbed the fatty tissues about the vessels at all. She will have no lymph or venous stasis from anything we have done. These axillary glands will be removed in two weeks from now if the tumor proves to be malignant. There is apparently no involvement of the lymphatics. In this case infection would be just as likely to cause enlargement of the glands as would metastasis from malignant disease. We will close the wound in the usual manner with silkworm-gut. A gutta-percha drain is inserted to permit the wound secretion to escape. It is taken out after seventy-two hours. There is no tension on the line of sutures, and still the skin is in much greater danger of necrosis than the average skin-flap, because there is so much fat here.

In these infections of the breast there is the same tendency to glandular involvement as in carcinoma. This is not the usual type of pus infection. When the bacteriologist makes a smear and culture from the breast, it will probably be negative, because the fluid in the breast is the lacteal fluid usually found in the chronically inflamed and irritated breast. The cicatricial tissue has obliterated the outlet of the lactiferous ducts, and therefore we have in these cases a lactiferous fluid—merely a fatty, creamy fluid, in place of pus.

That was one of the greatest evidences against this being a malignant lesion. Primarily it was not a malignant lesion. We are certain of that, because there were multiple foci and they were a considerable distance apart. That speaks against malignancy at once, and that formulates my opinion as to this being a chronic mastitis more than the mere presence of the fluid, because not infrequently you find that type of fluid present when there is a malignant lesion, and particularly a rapidly growing malignant lesion, and the younger the individual, the more likely is that to be the case. These lactiferous gland cells are active at this time of life, and, therefore, fatty degeneration and the secretion of milk are going on at the same time.

The additional danger of removing the tissues in the axilla is comparatively small, taken as a whole. The percentage of danger in the individual case following the removal of the axillary zone is over 80 per cent., the risk of amputation 0.8 per cent., the mere removal of the breast is practically no risk at all, the removal of the axillary glands with the breast has a small percentage of risk.

All the tension sutures which we have put in are silkworm-gut. The skin edges are coaptated with horsehair. We use horsehair in the skin because it is elastic; silkworm-gut is inelastic. Silkworm-gut cuts the skin; horsehair does not, Horsehair does not cut the skin because it stretches, but it will stretch only once. It does not rebound. If I stretch it again, it breaks. That is why it is ideal suture material for the skin. It is elastic, but it does not again contract; it gives without contracting. Silkworm-gut does not give any more than wire does. It produces skin necrosis, and when that occurs, you are very likely to get infection with saprophytes, so that the preservation of the margin of the skin is very important. In these cases we have to make such long flaps, and we have to remove the fascia and fat from beneath the skin, so that it has not been uncommon to have marginal necrosis, and it is not uncommon to get saprophytic infection after necrosis has taken place.

In these cases, if you do not provide for drainage of the secretions, which sometimes are considerable in amount, especially when there is much adipose tissue, as in this case, they accumulate and interfere with healing. Before agglutination of the surfaces occurs, before definitive adhesions can form, it is essential to provide for the removal of all of that secretion. Absorption in a tissue of that kind is extremely slow, therefore you hasten the consolidation of the scar by putting in a drainage-tube for forty-eight or seventy-two hours, thus allowing the wound secretion to escape. Then take out the drain. If you keep the drain in longer than seventy-two hours you are likely to get saprophytic infection, and then you will have secretion pouring out for twelve or fifteen days. You can open a joint, and if you will close it again, there will be no more tendency to ankylosis than if you had not touched it. If you put in a drain for seventy-two hours, or a week, or ten days, you have ankylosis in over 90 per cent. of the cases. If you follow the lines of these principles in surgery, you will find the same thread running through every strand, figuratively speaking. For a time we put in flap drains; that is to say, we used gutta-percha tissue without a wick. The wick acts as a siphon for a few hours only. After that time it acts as a plug. Rubber tissue around the wick makes drainage around the wick but not through it. The center of the drain is plugged. A strip of gutta-percha allows the fluid to escape when it gets under considerable tension. We want it to escape onto the dressing as it is formed, and, therefore, a large dressing is applied and fairly firm compression is put on the whole dressing, so as to keep the wound surfaces approximated as well as we can during the early stage after the operation. That approximation tends to lessen the hemorrhage. The approximation of muscle tends more to the lessening of hemorrhage probably than any other tissue, except when you come to renal tissue or adrenal tissue. This is beautifully illustrated by Sir Victor Horsley in his management of the oozing from the surface of the brain. He takes out a piece of the temporal muscle and applies it to the oozing area, and there is something coming from that muscle that acts as a hemostat. Just what it is has not yet been definitely settled. In operating on kidney tissue we find that hemostasis takes place quite rapidly. In renal surgery always open the kidney from the pelvic side. In going through the cortex you immediately have a great gush of blood and you think the patient is going to bleed to death in a few minutes, but just hold the kidney back and put on hand compression for two or three minutes, open the kidney, and there are not more than two or three vessels bleeding. The reason for that is the action of the renal cells. There is really no very great difference between renal and adrenal tissue, so far as the hemostatic effect of local compression is concerned. In making the approximation of the wound surfaces it is important to get accurate approximation, so as not to have primary effusion. The more tension you have the more likely there is to be oozing, yet the packing stops it.

As the axilla was not excavated in this case, there is no danger of a sponge being left in there. When the axilla is excavated, a sponge count is made, as in a laparotomy, because sponges have been left in the axilla a number of times; they have been left in the triangles of the neck, so that whenever we make a complete breast operation we always keep a record of the sponges just the same as in our laparotomies. This was merely a surface amputation, hence there was no need of keeping a count of sponges.

Let the record show that there was no count kept of the sponges; that the axilla was not cleansed. Let the record show further that there were numerous foci of cicatricial tissue an inch and a half apart, extending through the upper and outer half of the breast; that in these separate foci were cavities filled with a creamy, yellow, purulent-looking material; that around these cavities we found considerable infiltration of tissue; that in no place in the breast did we find definite, hard fibrous masses or a mass without the infected focus, resembling the macroscopic picture of a carcinoma. From the fact that there were multiple foci; from the fact that this patient is thirty-two years of age; and from the fact that there was no dry focus of infiltration, we believe that this is a mastitis with retention and possibly with infection. [This diagnosis was later confirmed by the microscopic examination. The wound healed per primam, and the patient made an uneventful recovery, leaving the hospital in perfect health three weeks after the operation.—Ed.]

RECURRENT OVARIAN CYSTOSARCOMA

HISTORY

Female, aged forty-six. Family and personal history negative. Menopause four years ago; has not flowed since.

Present Trouble.—Began four years ago (1908). Patient noticed that her clothes did not fit. Then noticed a distinct swelling of abdomen, more marked on left side, but gradually becoming general. This swelling continued to increase slowly in size, without pain or other discomfort, until about three months ago. At that time she noticed a hard mass about the size of her palm in the left lower quadrant of the abdomen, seemingly just inside the abdominal wall. It appeared to be fixed. It was not painful or tender, but increased rapidly in size. The general abdominal swelling also increased more rapidly, and patient began to have difficulty in breathing. Bowels have been becoming more and more constipated. Urine has always passed freely and nothing has been noticed in it. Patient lost ten pounds in weight in three months. Is growing steadily weaker. Has had no chill, night-sweats, or fever.

She was operated on in May, 1909, an ovarian tumor being removed, and after that felt well until the last Thursday in November, 1910. She was a pall-bearer at a funeral on a rainy day, and just during monthly period, which usually lasted three days. The flow stopped after the first day, and for the next two months she did not flow. Patient was up and around a week. Flowed February and March, 1910; flow was very slight in amount and lasted only one day. Since then she has not flowed at all. Has not felt entirely well since. Has a feeling of rawness internally over whole abdomen. Since operation patient has been feeling about abdomen for a possible return of the tumor, and the last day of June, 1911, she felt a tumor about the size of a dollar in the right lower quadrant of the abdomen. It was very hard, not painful, and would slip away from under her fingers. This has increased rapidly in size and has the same consistence as at onset, but has grown to such an extent that it entirely fills the abdomen and resembles a pregnancy. Since last operation patient has lost twenty-five pounds in weight and has also lost her appetite. The tumor was again removed. Says it was an ovarian tumor. The patient made an uneventful recovery. Bowels regular. No urinary symptoms.

May 7, 1912, patient returned to the hospital. Since her last operation, in August, 1911, patient has felt well. When she left the hospital she had been instructed to return every three months for reëxamination. May 3, 1912, patient was examined by Dr. Murphy and told to return for operation. She has had no subjective symptoms.

COMMENTS AND OPERATION

Dr. Murphy: What date was the first history written?

The Intern: 1908, when patient was forty-two years of age.

Dr. Murphy: This was at the first operation. How about the second operation?

The Intern: The second operation was done one year later.

Dr. Murphy: What I wish to know is about the pathologic findings in the first operation.

The Intern: It does not state in the report.

Dr. Murphy: The history of the pathologic findings should be there. I remember it very distinctly. We thought this an adherent cyst, but the pathologist said it was a cystosarcoma. The subsequent history shows where the pathologist helped us out at this time; it shows why this patient is having a recurrence. It was a mixed-cell sarcoma of the cystic variety. This time the tumor is much smaller than at any previous time. We hope that there are no adhesions to the intestine. She came in without any symptoms at all; came in on instructions to have repeated examinations made so as to be on guard before the tumor attained any great size. We will open the abdomen in the midline, between the umbilicus and the pubes. We are going through cicatricial tissue here, and are going through it with caution. On going through the peritoneum and entering the abdominal cavity, we find that there are adhesions present. They are the soft, friable adhesions one commonly sees in tuberculosis—the so-called "cobweb adhesions." There is a mass here that looks like an infected gland, but as there are no glands in that position, it is probably a secondary neoplasm. The bowel is fixed to the anterior abdominal wall. These are more of the cobweb type adhesions and are easily freed.

The ovarian cyst is now plainly visible. It is of a dark color. The patient is having metastases, and is in a more serious condition than she was in before. There is a neoplasm on the wall of the small bowel of quite a size. It is a secondary neoplasm. We must ascertain whether there are many of these before we attempt to do anything. There is also a larger cyst here. It appears to come well up into view, and is attached away over on the left side. There is another implant in that region, also of the sarcomatous type. The neck of the cyst is adherent to the anterior abdominal wall. It is going to be difficult to free this. The attachment to the anterior abdominal wall is probably the place from which this cyst springs—from the peritoneum.

I will try to remove the base of this tumor with its peritoneum. I am now examining the intestine to see what relation the colon bears to this tumor. The colon is free from neoplasm. When we get the line of demarcation between tumor and normal tissue established, we should be able to enucleate this with comparatively little trouble, and we should get into what is known as a bloodless zone.

I want to call attention to the physical characteristic of this neoplasm, namely, that it tends to maintain its cystic character all the time. It has not gone through the peritoneum. As far as we can determine now, it is just adherent to the serosa. We can easily remove the tumor. Just shell it out. On that you can base a positive diagnosis alone. You do not need to have a microscopic examination made to tell the story. Then put a puckering string of catgut around this abraded spot and turn it in.

The uterus is about normal in size. Now we must cover as much as we can this abraded surface of the peritoneum with the normal peritoneum of the opposite side. It is going to be a little difficult, still I think I can accomplish it because this woman is so very thin. We are able to take up the peritoneum and approximate it across the line and cover the abraded area fairly well, notwithstanding the fact that the abrasion of the parietal peritoneum was of considerable extent. However, in a stouter woman we would not have been able to do this.

We will close the peritoneum with a continuous catgut suture, and approximate the deeper structures by means of a figure-of-8 silkworm-gut suture, putting in three or four to insure a perfect result. The skin wound is closed with a continuous horsehair suture, dusted with bismuth subiodid powder, and sealed with collodion gauze. Over this is placed the usual dressing, held in place with the abdominal binder.

We were able to cover this raw surface better than I thought we would. The abraded area is fairly well protected with peritoneum on the inside, and there remains only a small zone for adhesions. This tumor will in all probability recur and go on to a fatal termination, possibly with greater rapidity than it did before the last operation, because it has attacked a new zone—the peritoneal surface.

This patient will be put on large doses of arsenic, hypodermically; that occasionally retards growths of this type, and occasionally retards them in a very striking manner. I will not say that I have seen a cure result, but I have seen large tumors disappear and then I have seen them reappear, but it gives the patient relief from pain because it stops the pressure of the growth.

The history of this case is the history of many of this class of cases, with one exception—the prolongation of life is away beyond the average period. We have succeeded in saving her life for a much greater period than is possible on the average. This cyst was not like any of the other cysts that one sees. If you take a section of this cyst-wall, you will find that it is not lined with epithelial cells, as is the case in all ordinary ovarian cysts and as you see it in sarcomatous conditions of the ovary.

This is a malignant hyperplasia of the connective-tissue elements, of the mesodermic element, and not of the ectoderm or endoderm cells, and it is for that reason that the tumor continues to recur and recur, but it does not recur in the gland itself. The recurrences were surface recurrences. The same thing occurs with implants due to ruptured ovarian sarcomata or ruptured papillomatous cysts. Papillomatous cysts cause the least numerous deposits of the peritoneal surface. When papillomatous cysts rupture, the peritoneum becomes studded all over with small tumors. In the last three months we have had three cases of this variety. In two of them we made some effort to remove the tumors. The case in which we made no effort to remove the growths has gone on to a fatal termination. In the two cases in which we made a partial effort at removal, notwithstanding the fact that the masses were not all removed, the effusions were relieved by thyroid and arsenic treatment. We gave large doses of arsenic, two and three grains at a time. Sodium cacodylate is the form of arsenic best tolerated by this class of patients. A few weeks ago we received a letter from a patient who some three years ago had had the same type of papillomatous cyst that could not be operated on. Everything was studded with tumors, but all the evidences of the disease have disappeared under the arsenic treatment.

We cannot say whether these tumors are malignant or non-malignant. We know that papillomata disappear on the surface of the skin (warts), a whole crop appearing and disappearing without any known cause. The same thing occurs in connection with papillomatous cysts in the peritoneal cavity. The microscopist reports all of them as being malignant. You can see why. Because the epithelial cells have passed beneath the basement-membrane. Clinically, we do not regard them as being carcinomata, and the fact that they disappear makes us want to include them in the non-malignant class of tumor. This, however, is not of that variety at all; it is a connective-tissue cyst formation, not an epithelial cyst formation. We know very little of this variety of sarcoma. We recognize its cells; we recognize its tendency to malignancy; we also know that it will appear in one ovary and after some months appear in the other ovary. We also know that it is the tendency of sarcoma of the ovary to become cystic. We do not know, however, why this metastasis occurs in the other ovary. We know that in sarcoma of one testicle the rule is for the other testicle to become involved, even when the one first involved is removed. If a patient has a sarcoma in one kidney, he always gets a second tumor in the other kidney. That is in refutation of the belief that these tumors are parasitic in origin. Why should these tumors come in both glands in just that way if they are parasitic in origin? The same is true of the infective variety of lesion. We know that when a patient has tuberculosis of one epididymis or of one testicle, he is in all probability going to get tuberculosis of the other epididymis or testicle, not as a primary lesion but always as a secondary lesion, showing that you can exclude these lesions from being parasitic or bacterial in origin.

RETROVERSION OF UTERUS

HISTORY

FEMALE, aged thirty-nine, married, has three healthy children. Was admitted to hospital because of lumbar and pelvic pains and extremely painful menstruation. Pregnant five times, the second and fourth pregnancy resulted in miscarriage at about the third month. The other pregnancies went to full term with normal deliveries.

In December, 1908, had an operation for acute left-sided pelvic pain, with vomiting, but no chills or fever. Previous to this, patient had not missed a menstrual period, but on close questioning she stated that the amount of flow and duration of the two periods preceding the operation were very much diminished. Two days preceding the operation she developed a sudden severe uterine hemorrhage, accompanied by pain and vomiting. This was independent of the menstrual flow. The abdomen was opened, but patient can throw no light on the pathology found or the nature of the operation performed. Since the birth of her first baby she has been troubled with severe lumbar pain, which obliges her to be in the reclining position most of the day. She has pain in the pelvis. There is no variation in the amount or duration of her menses in that time. She has no urinary symptoms.

COMMENTS AND OPERATION

DR. MURPHY: How old is she?

THE INTERN: Thirty-nine years.

DR. MURPHY: Her menstruation all her life, as you will note, has been painful and of the obstructive variety. Then she came to the period when she had to have an operation, the nature of which I do not know, by which the uterus was supposed to have been restored to position, but her condition has not been materially changed. She is still having her painful menstruation, and she has now a retroversion of the uterus of the third degree—the superlative degree. The uterus is resting in Douglas' pouch. She has some obstructive symptoms, and for that reason we are going to dilate the cervix and do a Pozzi operation on the cervix.

You will recall a paper read by Pozzi in this country in 1904, when he outlined the plan and reason for his operation for obstructive dysmenorrhea. Obstructive dysmenorrhea of the type mentioned is thought to be due to retention in the external os, and it is due to stenosis, first, of the external os; second, to dilatation of the cervix between the os externum and os internum. We have since that time had quite an experience with this operation, and it is one of the most gratifying operations we have, with little hazard and little inconvenience to the patient. It is so easily done. It consists, in the main, of producing a permanent dilatation of the os externum—the mere dilatation of the cervix does nothing. It contracts again and always seems to become smaller after dilatation than before.

You will remember in the early days of the American operation, so called, for hemorrhoids, we overdistended the rectum and the patients frequently got incontinence of feces. It is entirely different in the uterus. The uterus is a musculofibrous organ, and the fibrous tissue has a tendency to contract after it has been traumatized.

The patient is placed in position, the anterior lip of the cervix is grasped with a vulsellum, and the uterus drawn to the vulva. We will first dilate the cervix with forceps dilator. I have directed that dilator upward because the uterus is retroverted to the superlative degree. This is a temporary dilatation and is done with moderate force, as I am going to make a division of the cervix and do a typical cervical repair. Having secured full dilatation of the cervix with the various dilators which are usually used, we will proceed to curet the uterus. This curet is a dull curet, the kind we always use. Now you can get an idea of the size of this cervical canal. The cervix permits my finger to enter clear back to the first joint. It shows that this was a potent element in the production of the pain. It was an obstructive dysmenorrhea from an irritation of the os externum. Pozzi's suggestion for treatment consists in taking a wedge-shaped piece out of the muscularis of the cervix. The purpose of taking that out is to permit the approximation of the squamous epithelium on the outside of the cervix to the columnar epithelium on the inside, and thus produce a contraction of the cervical canal. When the two surfaces are united transversely with catgut you can never have re-formation of the divided portion of the cervix, and that insures a patent os externum. The os externum is always larger than normal. The operation is a simple one, easily done, and always gives relief. Two stitches are put in above and two below. This is just what we would do in a case of web-finger. If you leave the divided septum between the two fingers without suturing, the patient will come back with the web-finger returned as bad as before, if not worse; but if you connect the epithelium-lined surfaces in front and behind the web, there is no subsequent contraction. Two stitches are usually sufficient. That insures a permanent patency of the os externum.

To recapitulate: First I took out a V-shaped piece of muscularis from the cervix. That permits of approximation of the outer and inner surfaces of the mucosa, the squamous cells of the vaginal portion of the cervix, and the columnar cells of the endocervical mucosa. The stitches are put in so as to catch the columnar epithelial cells on the inner side, and the squamous epithelial cells on the outer side, bringing those into accurate apposition, so that there will be no possibility of a transverse healing. That is, the transverse incision is sutured longitudinally. Restoring that uterus to its normal elevation in the pelvis would not relieve this patient of her dysmenorrhea as long as this os externum remained closed as it was. It would simply relieve the pain in the back after menstruation, but would not relieve the first day's pain during menstruation. Ordinarily, however, this is the only operation that these patients need to have to get relief. The uterus is allowed to recede, and the vagina is packed with sterile gauze strips.

In the evolution of the treatment of retroversion we have had many discussions and many differences of opinion, and the result has been that we have had many operations devised and performed. How is a retroversion evolved? How does a retroversion occur? In a retroversion of the uterus, occurring either suddenly or gradually, it always occurs with an inflammatory condition in the pelvis which finally draws the uterus into Douglas' pouch and fixes it there, though in the enormous percentage of cases it occurs without fixation or adhesion in Douglas' pouch. The weight of the uterus and the pelvic pressure are all carried by the cervicocorporeal portion of the broad ligament. This is what keeps the uterus from descending. Intra-abdominal pressure on the uterus in the normal position is brought to bear on the posterior surface of the uterus, pressing it forward toward the bladder. As the retroversion occurs the uterus tilts backward more and more, and the round ligament becomes longer and longer, until finally there occurs the superlative degree of retroversion. The intra-abdominal pressure is now on the anterior surface of the uterus and the round ligament—that is, the balancing rope or cord of the uterus—passes in front of the cervix, but if it is torn from its attachment and passes behind the line of the cervicocorporeal attachment of the broad ligament, it is a restraining ligament rather than a supporting ligament, and it holds the uterus backward in place of holding it forward.

In the treatment of retroversion you have to reëstablish the anterior supporting ligament, which is what? The round ligament. Unless that can be reëstablished in its normal relation one has a failure in results. Wherever one attempts to do this with adhesions in the pelvis, nothing but failure can result. It has then been attempted, under unfavorable conditions and under conditions which our knowledge of mechanics as well as of pathology did not justify; that is why the Alexander operation has gradually come into disrepute.

For a number of years we did the ventral fixation operation for retroversion until we learned that that was not the best procedure for retroversion: that it is mechanically incorrect. We will now place the patient in the dorsal recumbent position and open the abdomen so as to correct the position of the uterus. The incision is made in the median line, between umbilicus and pubes. I do not know what adhesions exist here from the previous operation. Something appears to be adherent beneath the peritoneum, so I will open that with greater caution. When incising the peritoneum, never cut between the two forceps that grasp it, but always cut on the slant, or incline. The air rushes into the abdominal cavity, and that protects one against accident or division of the omentum or of the intestine. By cutting between the forceps we may produce serious trouble. The wound edges are retracted so as to give us better access to the uterus.

I will now endeavor to get the fundus of the uterus up out of the pelvis. I can feel the cervix pressing through the anterior vaginal wall, and back of that I feel a little fibroma which I did not recognize in the original examination. I am trying to catch the broad ligament. I have succeeded in getting my finger under the broad ligament. I have the heavy fundus that was down in the culdesac. There is also a fibroma in this fundus. There is a fibroma which I thought was in front of the cervix. I have pulled free the adhesion that existed. This patient is a nullipara, and I am not at liberty to take out the uterus, but I will enucleate the fibromata, which is the only thing I am justified in doing. This accentuates, more than the ordinary case, the significance of the downward displacement of the round ligament. The round ligament is down almost to the cervix.

Now, doctor, I want you to read again about the duration of her menstruation.

[The intern reads history.]

This will be just an enucleation—a myomectomy. I will cut through the capsule of the fibroma and enucleate it. These fibroids have in no way interfered with her menstruation. In fact, it is the opposite. In place of menstruation increasing in frequency and increasing in quantity, the menstruation has diminished in quantity and diminished in frequency—is that right?

The Intern: Diminished in frequency, but not in quantity, doctor.

Dr. Murphy: Diminished in frequency, but not in quantity? Thank you, doctor. You will remember, in the many discussions we have had here concerning fibroma, that one of the things that we have endeavored to accentuate has been that menstruation is influenced by fibroids in proportion to the relation of the fibroid to the mucosa. These fibroids are subserous, and subserous fibroids do not influence menstruation at all. These are striking examples of that situation as far as these lower fibroids are concerned.

We will make an entropium of this serosa, so as to avoid having any abraded or raw surface, which might lead to the formation of adhesions. In the hemorrhage from fibroma you have to take into consideration not so much the size of the fibroma as the relation of the fibroma to the mucosa. A fibroma not larger than a filbert situated in a submucous position—a submucous polyp, if you please—influences menstruation more than a fibroma six inches in diameter situated subserously.

There is still another fibroma below. Let us see what relation that bears to the trouble. It appears to be deep in the muscularis. There is practically no bleeding at all from that as I enucleate it. I am going to turn in the cut edges of this serosa as I did in the case of the other tumor. Catgut is used for this purpose.

The next step in the operation will be to throw this uterus forward. You can see why any external operation would have been a failure here. The question arises whether this fibroid was present at the previous operation. There is no record of it and no mention of it by the patient. She did not mention it to me and she did not mention it to the doctor who took the history.

I do not wish to leave a raw surface exposed on the fundus of this uterus; if we do, we will have an adhesion to it and that is not desirable at all.

This woman is still in the child-bearing period. We will take off this vulsellum which has held up the uterus and let the uterus come around to its normal position and close up the wound, from which we have a little oozing as a result of the attachment of the forceps.

The next step is fixing that uterus so it will stay forward. Let us see where these round ligaments are. They are far below the dome of the fundus. We will bring those round ligaments well over so as to tack them with catgut sutures to the highest portion of the dome and behind. I throw the uterus forward toward the bladder, where it belongs, and I will fix the round ligaments behind, on the posterior surface of the uterus, so that they will hold it in proper place. Then, you see, each round ligament passes directly upward and outward to the internal opening of the inguinal ring. I am not disturbing the relationship of the tube at all. This patient can become pregnant and that round ligament will elongate, as it should. The round ligament carries none of the weight of the body of the uterus. It merely balances it. The intra-abdominal pressure will hold it in place. I will have to shorten it about an inch and a half. I want to call your attention to the descensus of attachment which has taken place, which is a significant thing. The normal descensus takes place when this patient is changed to the sitting position. She has this rope or ligament fixed to the highest point up above the cornu, and that gives the greatest possible leverage advantage one can get from the muscle from this side. Each round ligament is looped behind the uterus, tacked to the fundus with a few catgut sutures, and then two sutures unite the loops to each other. That is ample to support the uterus subsequently.

Now comes the closure of the abdomen. I make an ectropium of the cut edges of the peritoneum by turning them out. I wish to prevent the cut edges of this peritoneum from being exposed on the inner side, because if they are exposed we will have adhesions within at the line of division. We have done that now for ten years as a routine procedure. We have had many opportunities to reëxamine these patients and we have been impressed by the absence of organic adhesions at the line of union, compared with what we had before we made the ectropium. We believe it is a very material aid. There is no abraded surface there to which adhesions can take place. So important is that in the avoidance of traction following operations that we have come to consider that advancement in the next ten years must be on the line of avoidance of organic adhesions of the omentum and intestines to the anterior abdominal wall. The intestinal adhesion that occurs is the most serious one, because you have the intestine attached behind by its mesentery to the spinal column from the second lumbar. We close the peritoneum with a continuous catgut suture.

The next step is the accurate closure of the aponeurosis of the rectus. We will not include in the suture the muscles nor the tissues external to the aponeurosis, because we have learned, from our knowledge of histology, that if you let some muscle or fatty tissue come in between the fibers of the aponeurosis, you have failure of edge-to-edge union and a tendency to spread, with the subsequent development of hernia. The rectus gives support in a perpendicular direction and little support in a transverse direction, so it is important to get accurate apposition of the aponeurosis of the rectus because that is the great transverse support against the formation of hernia. We again use catgut. The skin and fascia are approximated with figure-of-8 silkworm-gut sutures. The skin edges are carefully coaptated and united with horsehair.

Take the history of the case—obstructive dysmenorrhea, pain in the back, discomfort in the pelvis, without any material increase in the quantity of the flow. After the abdomen was opened we found a superlative degree of retroversion. Study of the case further showed that, as that retroversion evolved, the round ligament slid further and further down in its attachment. Shortening of that round ligament by the Alexander operation would not have done this patient one particle of good.

In the cure of retroversion there are two plans of procedure. If you have a retroversion with descensus of the uterus, that is, with prolapsus of the first or second degree, then you have to do some operation which either fixes the uterus to the anterior abdominal wall or you have to take out the uterus. When you take out the uterus and do a complete hysterectomy and shorten the transverse ligament, in a considerable percentage of cases you effect a cure. When you take out the uterus, however, in other cases, you have finally prolapsus of the vaginal wall and prolapsus of the rectum and prolapsus of the bladder, and you have nothing left to which to anchor these structures subsequently. In an ordinary retroversion in a nullipara without enlargement of the uterus and without pelvic inflammation the Alexander operation is ideal. The operation as we have done it here for eight years is folding the round ligament over the dome of the uterus, suturing it there. This shortens the round ligament and tips the uterus forward.

The wound will now be dusted with bismuth subiodid and sealed with collodion gauze. The stitches will be taken out in about ten days. A large abdominal pad and binder make all snug.

RECTOCELE AND PERINEAL LACERATION

HISTORY

HOUSEWIFE, forty-eight years old, entered hospital for the repair of a rectocele and lacerated perineum. She was married at the age of nineteen and has given birth to six children. She has had three miscarriages. Aside from trouble connected with childbirth she has been free from illnesses of a serious nature.

After the birth of her first child, twenty-nine years ago, her perineum was torn badly. One year later she had a miscarriage at the third month, due to a fall downstairs. Following this the uterus came down and protruded from the vulva at least two inches, she thinks. A doctor applied a tight pad and binder and treated her for weeks. The uterus never came so low again, though she was often bothered with it. She wore a pessary for six months following the birth of the fourth child, fifteen years ago. This seemed to be of value in keeping the uterus in place. After the birth of her fifth child a doctor told her that her rectum protruded into the vagina badly. For twelve years she has not noticed the uterus abnormally low in the pelvis and has not had the dull, dragging pains that she formerly had in such a marked degree. Her bowels have been sluggish all her life, but much more so since her fifth child was born. During the past three years they have been very sluggish and she never has a movement without catharsis or the use of an enema. Temperature and urine normal.

COMMENTS AND OPERATION

DR. MURPHY: What is the matter with the woman?

THE INTERN: That history does not tell what is the matter with her.

DR. MURPHY: Important facts are left out. First, when she was examined by a doctor some three years ago he found her uterus was low. Since then she has reached the menopause, and since the menopause she has had atrophy of the uterus, so that now it is not larger than my thumb, and it is up in fairly good position. She does not have procidentia uteri. The uterus is in an anteflexed position. When she goes to stool the rectum bulges out of the vagina. She has a laceration of the perineum, with prolapse of the rectum. There is no prolapse of the anterior wall of the vagina. She has no vesicocele and no procidentia.

In considering these cases the other day we divided the treatment into four kinds: first, cure by repair of the perineum, with reproduction of anteversion. Second, ventrofixation as a part of the treatment. Third, implantation into the anterior abdominal wall, and, fourth, extraventral splitting of the uterus so as to hold the vagina and rectum in place. The last three methods cannot be considered in this case, because it is only the prolapse of the rectum that is troubling her. She wants repair of the levator ani muscles and closure of the large vaginal opening below, so that when she strains she can have stool without the anterior wall of the rectum coming down between the labia. That tells the story. The history should tell this. The patient has nocturnal urination. What does this patient want to be relieved of? She wants to be relieved of the projecting forward of the posterior wall of the vagina and the anterior wall of the rectum. That tells the story. [The patient is in the lithotomy position.] Look at the rectum, coming right out without any straining at all. When she strains, the rectum protrudes more than it does now. There is no vesical prolapse at all.

We must expose the levator ani muscles higher up, in order to get them fixed. If you do an ordinary perineorrhaphy in a case like this, you would not get the desired result. If you do the old-fashioned operation of repairing skin and mucosa, the result will be much better. She has a very good perineum now, just as good as some women get sometimes from a perineorrhaphy. Lawson Tait is the man who really put perineorrhaphy on the proper basis. What we want to do here is to restore the muscular support.

We will make a U-shaped incision at the introitus vaginæ, encircling it and extending from caruncle to caruncle on either side to where the normal vaginal opening should be. I will then burrow upward under the vaginal mucosa until I reach the levator ani muscle. I want to keep under the mucosa and on the muscularis; I spread my scissors so as to separate the fascia without danger of producing a severe hemorrhage. The gauze-covered finger is also very useful.

VISITING PHYSICIAN: How many stitches do you put in the levator ani muscle?

DR. MURPHY: About five, as a rule.

VISITING PHYSICIAN: Do you use the figure-of-8 stitch?

DR. MURPHY: No, not at all. Now I come to the rectal sphincter and I pick that up. The sphincter ani has not been divided. We will now unite the torn levator ani muscles and fascia with catgut so as to give her a firm perineal body, which will also prevent the rectum from bulging through the vaginal wall. We have in all our stitches to support the perineum proper. We will then put in a loose subcutaneous and submucous stitch that goes across from one side to the other just beneath the vaginal mucosa. The vaginal mucosa will fall in over that stitch and bury it. This is essentially a Tait operation. It is a muscular approximation. You get these two levator ani muscles coming together. There is the real perineum. I will pass these stitches through the under portion of the anterior vaginal flap or the inner portion of the mucous flap of the posterior wall of the vagina for the purpose of obliterating that dead space in which occasionally a clot forms and which acts as a decided retardation to union. There is accurate apposition of the full length of the levator ani muscle clear down to the central raphé. The next thing is to put in a subcutaneous stitch. The perineum is now an inch and a half thick. If that holds,—and we have every reason to believe it will,—we will have a good and serviceable perineum, and there will be no further descensus of the anterior wall of the rectum. That is a submucous stitch, and finally, as we go a little farther down, it becomes a subcutaneous stitch.

This patient will receive lysol vaginal douches twice a day. When she wishes to urinate a soft catheter will be used and after each urination she will have the perineum washed off with lysol. The only thing which will interfere with perfect union is sepsis; therefore we must guard against that.

[The final result in this case was all that could be wished for. —Ed.]

UNUNITED FRACTURE, SHAFT OF RIGHT HUMERUS

HISTORY

MALE, railway brakeman, aged twenty-seven. Entered hospital because of an ununited fracture of shaft of right humerus. Denies syphilis in both self and family.

February 5, 1908, he was riding in the caboose of a freight train when a rear-end collision occurred. The car in which he was riding was completely demolished. He was rendered unconscious and remained so for fully three days. He received many injuries, all of which healed without incident except what he says was a compound fracture of the shaft of the right humerus. Two weeks after the accident his arm was taken from the wire splint in which it had been placed. The patient is not sure that the discharge had ceased, but knows that there was never much drainage. At this time the fragments were wired. Six weeks later the bone was still ununited. Wire was removed and he carried his arm in a cast. Three months after the accident he was operated on again. He thinks the callus was cut away and bone-ends were united with catgut. This was four years ago, and a false point of motion still remains at lower third of the right humerus.

Temperature, normal. Urine, normal. Hemoglobin, 80 per cent. Leukocyte count, 10,000.

COMMENTS AND OPERATION

DR. MURPHY: This man gave me to understand that there was a fragment of wire in there. He told me they took out a part of the wire, but did not take out the other part. Here is a typical pseudo-arthrosis with a good lining for the joint. Nature made a good artificial joint. We have been learning to make them where we need them—in the knee, elbow, hip, etc. The fracture was when?

The Intern: February 5, 1908, four years ago.

Dr. Murphy: How many operations has he had?

The Intern: Three.

Dr. Murphy: It was a simple fracture to begin with; it does not look in the skiagram as though it were a compound fracture. What was the original treatment?

The Intern: It was put in a wire splint and left for nine days.

Dr. Murphy: What then?

The Intern: It healed completely in two weeks.

Dr. Murphy: We have pointed out repeatedly in our teaching that the reason the bone-plate and mechanical internal appliances are going into disuse is because they are put in open compound wounds. They must not be put in until such wounds have entirely healed and are aseptic beyond question. Every practitioner seems to be putting in a metal bone-plate. The steel plate of Lane must be put in with brains, and it must be put in under proper aseptic precautions, and it must be put in a field that is absolutely aseptic beyond question. That is what is happening with Lane's metal plate; that is what is going to happen with the bone-plate because to keep it aseptic is far more difficult than is the case with the Lane plate. I know of at least six cases in which the Lane plates had to be removed. Why? Because in all six of the cases the plates were put in under improper conditions. The failure is not to be charged to the Lane plate, but to the practitioners who undertake to do this work without getting all the facts and reading carefully between the lines, and who do not know how to do the work in an aseptic manner, after they have read the lines and between the lines. Even then we are apt to fail sometimes. Let us keep that in mind. Lane's plate was never intended to be inserted in a compound fracture until the wound had entirely healed and was altogether aseptic. Neither wires nor nails are to be put in in cases of compound fracture, because they have to be taken out, and the great majority

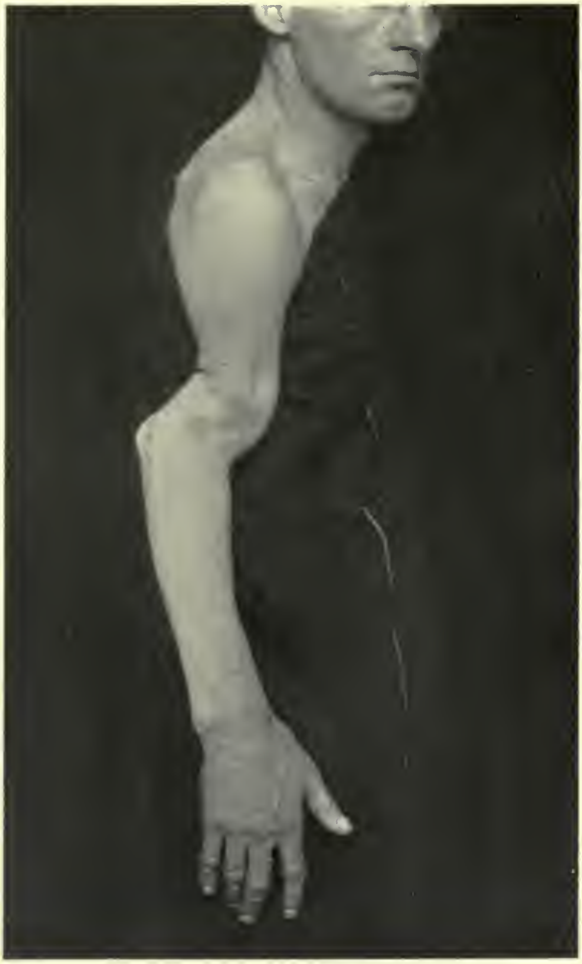

Fig. 124.—Photograph showing position of arm on attempted flexion of the elbow-joint. A typical false joint was formed at the seat of fracture in the lower third of the humerus.

have to be taken out before there is union. That has been our clinical experience, which has been enormous.

Regardless of what the etiologic factors were, non-union is likely to result if a plate or other foreign body is put in when the indications are not suitable for it. The nearer one is to the elbow-joint, the more unpromising the possibility of union is, because it is practically impossible to immobilize that elbow-joint at that particular point and prevent the brachialis anticus from contracting and prevent the extensor and flexor muscles of the forearm, that are attached to the condyles and condyloid ridge, from contracting and pulling that downward. When you straighten the forearm, see what happens! The brachialis anticus and the flexors and extensors of the forearm pull the lower fragment clear around at right angles. That is the difficulty we encounter in these cases. The implant in this case has been worked out of the lower fragment and pushed up into the upper fragment.

It was a bovine implant, and does not perform the function that an implant from the patient's own bone does. The capillary vessels of the human bone never pass into the Haversian vessels of the bovine or animal bone and carry with them the osteoblasts necessary for the regeneration of bone. We have learned that by experience. We did not know it when this method of splinting was first proposed, but at the same time we thought that it would give the mechanical support necessary, as ivory does, and that we would get union around the implant, although the implant acted as dead aseptic material.

In operating on this man now we are going to use an implant from his own tibia, which is the proper thing to do. We always take an implant from the same individual, and the best material to take is the compact bony tissue of the crest of the tibia. The crest of the tibia gives you the greatest latitude. Here we must put in a long implant that will go away down in the lower fragment, so that it will mechanically counteract the contraction of the muscles of the forearm from the moment it is put in, and it must be strong enough. I will make a magnesium metal nail which I may need in this case later. Magnesium is absorbable, and as it absorbs it produces a considerable quantity of gas, so that it blows up and balloons the inner lining of the wound. You may need to aspirate and remove it if the accumulation of gas gets too large. You can hasten the absorption by passing through this wound any acid solution, but the rule is that it absorbs quickly without such aid. The tissue fluids absorb the magnesium very well.

Our plan of procedure in this case will be, first, to expose the fragments and place them in proper position. There are fixation and shortening of the anterior elbow-joint capsule. In making our incision, what have we to bear in mind? We have to remember the musculospiral nerve. I will make my incision in front, right at the margin of the biceps muscle. I am going near the anterior incision that was made here in the previous operation, so as to get near the same line of injury to the subcutaneous tissues. The upper end of the lower fragment is thus easily exposed. A beautiful capsule has developed around this false joint, showing how nature does the work. How nicely developed the capsule is! It is like the normal capsule of a joint, although this is a pseudo-arthrosis. There is fibrous union of the fragments. We will liberate the capsule from the fragments so that the bone-ends will be exposed freely. The anatomic relations are essentially destroyed, and what we have to do is to keep close on the periosteum all the time, so as to keep out of trouble. It is these new ligaments that have developed in this pseudo-arthrosis that are giving me the trouble now.

The cartilaginous surface of the new joint is fairly well developed. We must take all that out because we want to get accurate contacting of bone to bone in order to get bony union. The fragments come in accurate apposition without difficulty. I will remove the cartilaginous covering of the lower fragment and of the upper fragment so as to have a raw surface of bone for an end-to-end union.

We are now ready to get the bone implant, which we will take from the crest of the tibia in the usual manner. The fragment will go into the medullary canal of the upper fragment quite a distance—an inch and a half or two inches. We will enlarge the

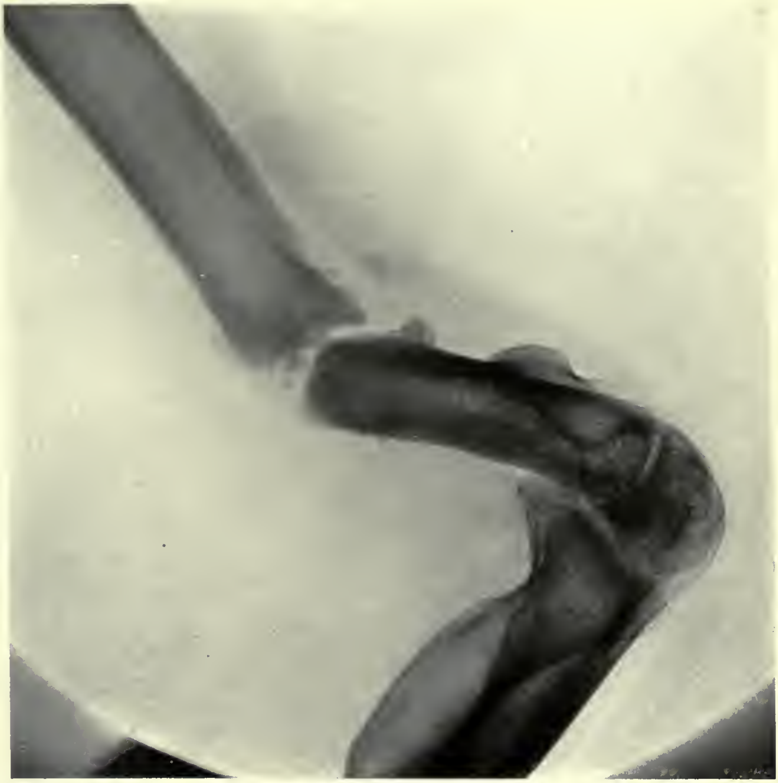

Fig. 125.—Radiogram showing lateral view with lower fragment tipped forward above. Ends of both fragments are covered with an abundant callus, but with no attempt at union. Typical pseudo-arthrosis.

canal with our medulla reamer so as to receive the implant. The lower fragment will also be reamed out, but for a shorter distance than the upper.

In taking off this piece of tibia we never make the incision over the compact bone, but over the tibialis anticus, so that the line of scar will not be over the compact bony tissue; then we go in through the tibialis anticus onto the periosteum. We do not know that that periosteum has any value. In fact, from the pictures we have had, it looks as though it is a detriment at this age. In children it is an advantage.

I will remove a piece of bone about four inches long, and 5/8 by 3/8 inch thick and wide. A small drill hole above and below will mark the length of the implant. That guides my instrument in cutting the bone. The retractor guide which I devised will guide my chisel in cutting out the piece of bone and also keep the soft tissues away from the chisel, so that they will not be injured. This retractor guide has two short prongs, one on either end. These two prongs go into the drill-holes I have made and hold all the tissues back out of my field. I am going to saw the bone crosswise at the point where I made the drill-holes, because that is where I intend to fracture it. I take out a V-shaped piece of bone all along the edge of the retractor and then the bone will fracture in that line. If we hurry, without taking out a sufficient V, we may fracture the fragment crosswise. That deficiency in the tibia fills up readily. The implant is held with forceps, and when removed is carefully placed in a piece of sterile gauze so that it will not be touched by hands or instruments and remain aseptic. We have the fragment ready to be inserted.

The incision in the leg is closed with deep catgut sutures, the muscle being sutured over the trough left in the tibia after the removal of the implant. The skin is sutured with horsehair. The wound is dusted with bismuth subiodid, a desiccating powder, and sealed with gauze saturated with collodion.

Let the records show that we took out a piece of bone four inches long by five-eighths by three-eighths, triangular in shape. In making the incision we came down on the musculospiral nerve, displaced one-half inch inward by contraction of the cicatricial tissue. We were sure it was the musculospiral nerve. We kept it in view all the time during operation so as not to injure it. After taking off the capsule of the pseudo-arthrosis, which capsule was firm, compact, and hard, resembling the normal capsule of the joint with synovial membrane (cicatricial), the ends of the fragments were exposed, both covered with cartilage. The cartilage was entirely removed and the ends of the bones freshened. Then with the reamer a hole two inches long was made in the medullary canal of the upper fragment, and another an inch and a half long in the lower fragment. The ends of the fragment of the tibia were rounded off to fit into these holes in the fragments. One end of the implant was driven into the upper fragment, and with a little careful manipulation it was finally, with as little tension on the lower fragment as possible, adapted and slid into the lower fragment; then it was driven back into the lower fragment by a rectangular bone-crushing forceps which grasped the implant on the side. This implant establishes a firm union of the two fragments and will play its rôle in the regeneration of bone, which will proceed from the upper and lower fragments through the implant. The implant is finally absorbed.

A test made of the musculospiral nerve with the faradic current at the completion of the operation showed the nerve intact and not disturbed by the operation.

The deeper structures are united with catgut. The skin is closed with horsehair. The arm will be put in a plaster-of-Paris cast reaching up over the shoulder. The arm is flexed and fixed at right angles in front of the body. The cast extends well down on the wrist and firmly up against the shoulder, including the shoulder, that is, over the cap of the shoulder, clear up into the neck. The arm must be kept in that position for seven weeks, perfectly still, until the bone fragment is filled with capillary vessels from above and below, and until the new bone spans the gap across the line of separation. First, there is a revascularization of the Haversian canals in the implant. That is, they are filled with Haversian vessels from the upper and lower fragments, which carry osteoblasts and osteoclasts through the

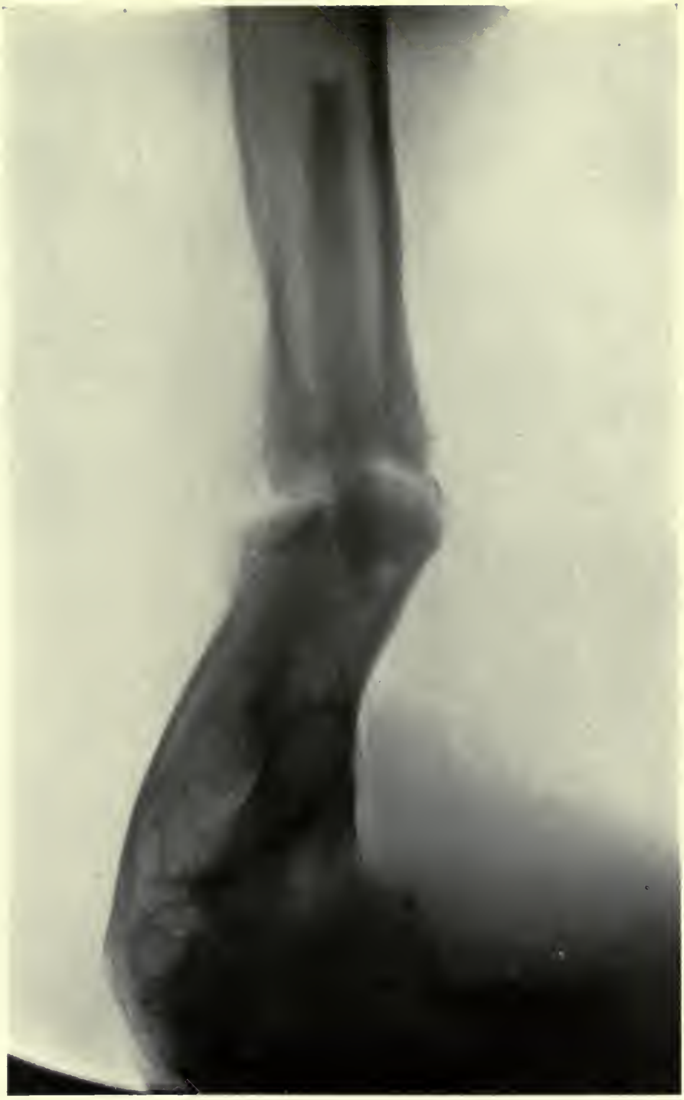

Fig. 126.—Radiogram taken five months after transplantation of piece of tibia into humerus. Firm bony union with regeneration of bone proceeding from both upper and lower fragments. Patient has full use of arm, although complete extension is still impossible.

Haversian canals in the implant. Finally, as bone regeneration progresses there will be in the center living bone with a shell of dead bone outside, and you have a bony union long before the dead piece of bone is removed by absorption, but all dead bone is finally taken away. These Haversian loops anastomose, and the new lamellæ are incased in the old lamellæ. The osteoclasts take out a brick or cell, and the osteoblasts put in a new one. The only rôle which the implant plays is that of a scaffolding for the Haversian vessels with their osteoblasts and osteoclasts.

[The progress of this case was uneventful and finally terminated in complete bony union and an arm anatomically and functionally perfect. Bone regeneration progressed rapidly, as shown in the skiagram.—Ed.]

OSTEITIS FIBROSA CYSTICA OF RIGHT HUMERUS

HISTORY

School-boy, thirteen years old, entered Mercy Hospital because of lameness and tenderness of right upper arm. His family history is negative. He was always free from digestive disorders of all kinds; never had rickets, scarlet fever, rheumatism, tonsillitis, or sore throat. He always was a strong, active boy, with full use of both arms, and both arms and shoulders were of the same conformation.

Early in 1911, while throwing a ball very hard, he experienced a sudden pain in the right arm, four inches below the shoulder. On attempting to raise the arm he found that he could not do so; it hung limply by his side. A doctor told him that it was broken, and placed the arm in a splint for six weeks. Union was apparently perfect, and he gradually regained full use of the arm.

In March, 1911, he fell on a stairway, striking on his right arm and breaking it. He carried the arm in a splint for six weeks, and again it became perfectly useful, but for four or five days only. He fell to the floor, striking on his left shoulder, but the right arm broke (third fracture). There was little pain connected with the injury. The arm became useful again, though it remained a little lame.

In July, 1911, he attempted to sit down on the floor and felt his arm and hand were limp. He had no pain. The arm was not fractured, but on examination he noticed for the first time that the arm was larger at point of fracture and uneven. All the fractures had occurred at the same point. Arm again became useful. Four weeks ago arm was lame and he could not raise it from body, and a tenderness has been present at site of fracture since that time. Wassermann negative. Leukocytes,

8200. Hemoglobin, 80 per cent. Urine alkaline, but otherwise normal.

COMMENTS AND OPERATION BY DR. JOHN F. GOLDEN

Incision was made directly over point of right shoulder on its outer and slightly on its anterior aspect. This divided the deltoid longitudinally and passed down to the bone, the shaft of the humerus, for fully six inches. At the upper angle of the incision the capsule of the joint was exposed, and at the lower angle could be seen the musculospiral nerve. This nerve was tested with the excitor both before and at the completion of the operation and found to be uninjured.

At a point 5¾ inches below the head of humerus the shaft showed absolutely no signs of involvement. Here the bone was freed of all its attachments, retracting the musculospiral nerve. A chain-saw was placed about the humerus at this point and the bone was divided.

The capsule of the joint was now divided circularly at its attachment to the neck of the humerus. The muscular attachments about the head were freed, keeping very close to the bone and even removing a small amount of it at the greater and lesser tuberosities. The outer head of the biceps was lifted from its place in the bicipital groove and displaced inward. It was not cut.

Now the upper end of the humerus was plainly visible. This portion was fully 5¾ inches in length and triangular in form, with broad end at upper end of bone. The largest diameter was 2½ inches. The mass was globular, expansible, and cystic, with eggshell-like portions of bone in places, and brittle. In other places it was more fibrous and less fragile. The periosteum had previously been removed when the muscles were taken off. Periosteum was carefully examined and found to be free from involvement in the process. Beginning at the inner side, the periosteum was elevated and the mass rolled outward and upward, detaching the periosteum from it until it was completely freed. Great care was used lest the musculospiral nerve be endangered in this process. The head of the humerus was not involved

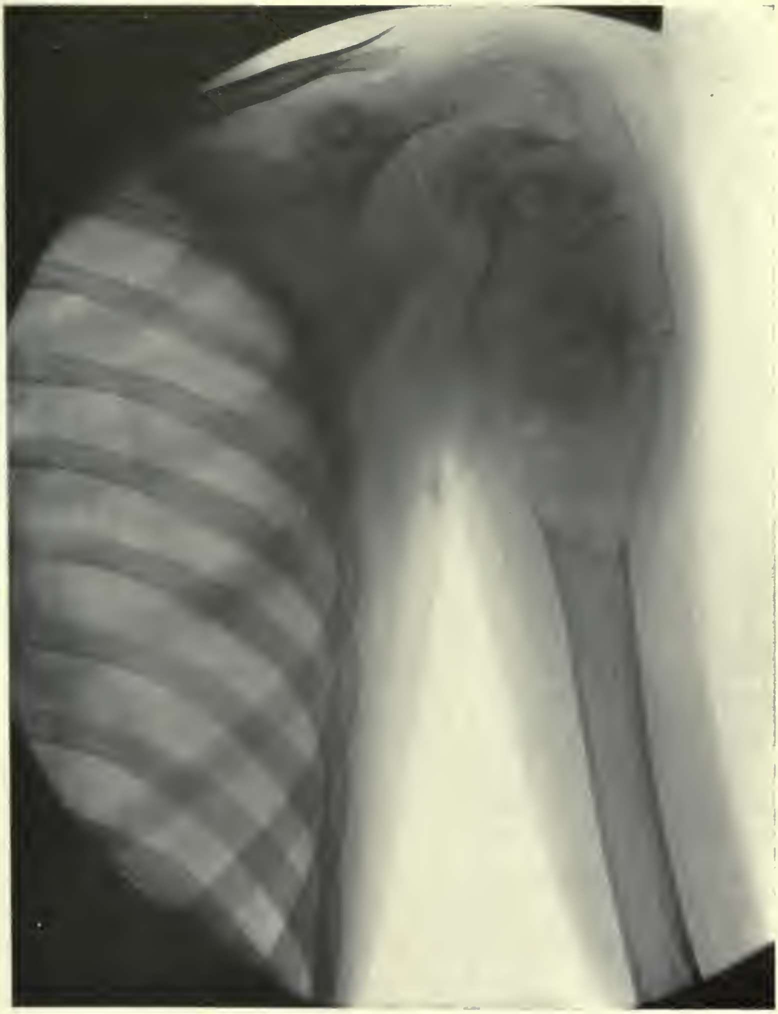

Fig. 127.—Skiagram showing case of osteitis fibrosa cystica of upper extremity of humerus before operation.

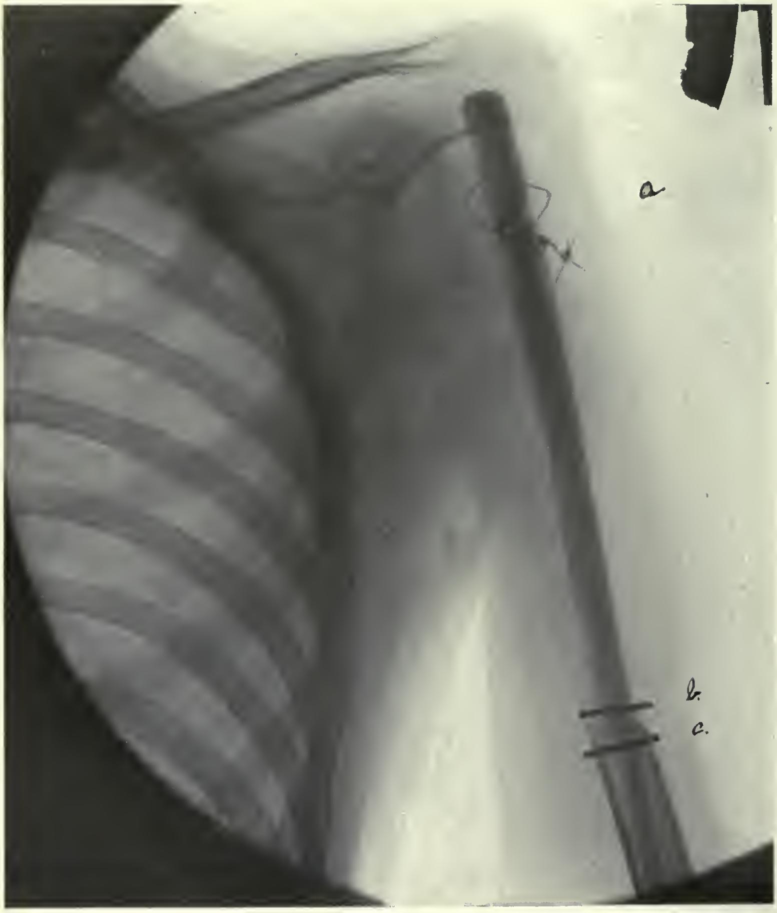

Fig. 128.—Skiagram showing tumor removed and tibial transplant in place. The lower end is inserted into the medulla of the humerus shaft and held by two nails driven through both bones. The upper end is in the glenoid cavity. The muscles are sutured around the transplant with phosphor bronze wire.

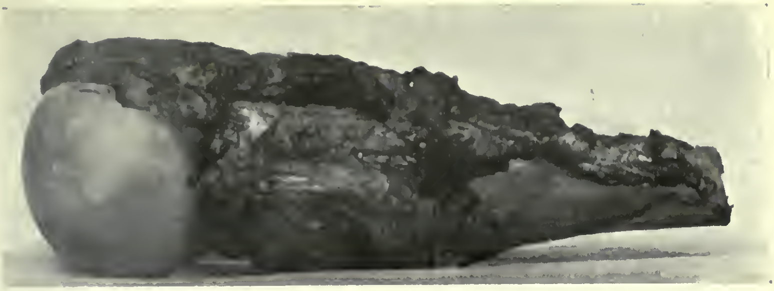

Fig. 129.—Photograph of removed diseased bone, showing cystic condition of bone above and normal shaft below, where musculospiral nerve crossed (view 1).

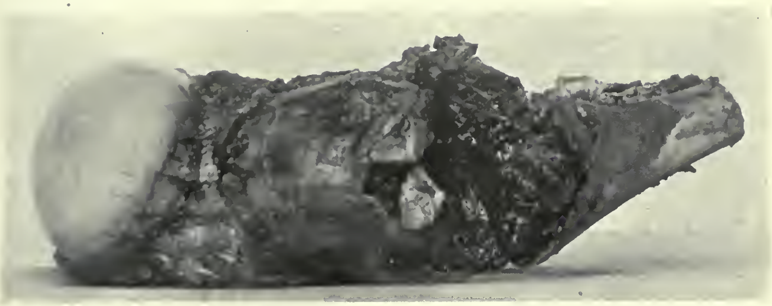

Fig. 130.—Photograph of removed diseased bone, showing cystic condition of bone with wall purposely broken in to demonstrate shell (view 2).

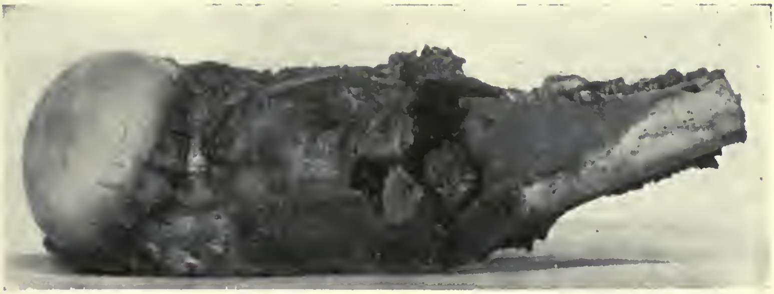

Fig. 131.—Photograph of removed diseased bone, showing same as view 2.

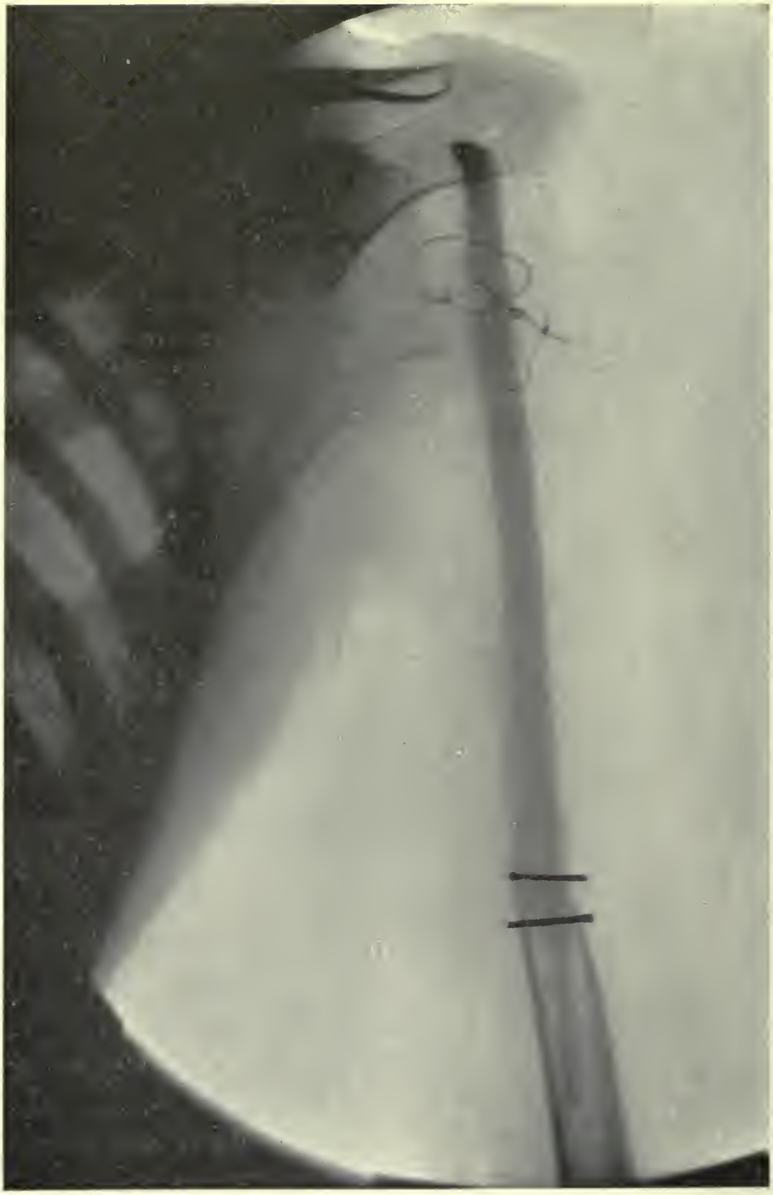

Fig. 132.—Skiagram of transplant five months after operation, showing regeneration of bone progressing upward toward joint from shaft of humerus. The new bone is easily visible along the edges of the transplant.

in the process, and the articular surface was apparently perfectly normal.

It was a question whether or not to remove this head. It finally was decided to remove it.

A transplant seven inches in length was now removed from the right tibia and ends rounded into shape. A hole was drilled in each end of this strip, the lower hole being about 1½ inches from the end. A hole was also drilled in the medulla of the shaft of the humerus, and the transplant inserted into this hole a distance of 1½ inches. A nail was placed transversely through transplant and humerus, thus preventing the transplant becoming further embedded in the medullary canal. Using a catgut suture as a purse-string, the capsule of joint was puckered in about the upper end of the transplant, which rested in the glenoid cavity. Now two wire sutures were passed through a hole in the upper end of the transplant and also through the joint capsule. Muscular attachments were not included in this. The wires were tied about the capsule only as an additional means of keeping the transplant in position in the glenoid fossa. The old periosteum of the humerus was sutured around the transplant. This carried the muscular attachments, none of which had been separated from it. A row of sutures closed the incisions in the fascia and aponeurosis; another of horsehair closed the skin incision. A plaster cast was applied, covering the arm from the side of the neck to the wrist, with arm at right angles.

[*Note.*—Six months after operation: Patient made perfect recovery and has now full function of arm and a movable shoulder-joint. The improvement is very satisfactory. The regeneration of the upper end of the humerus is progressing well, proceeding upward from the shaft of the bone.—Ed.]

ANKYLOSIS OF LEFT ELBOW

HISTORY

MALE, aged thirty-one, single, salesman. Came to hospital because of ankylosis of left elbow. Childhood disease with good recoveries. Typhoid fifteen years ago, also with good recovery. No tonsillitis, alveolar abscess, discharging ears, or enlarged glands in neck. Patient has been in perfect health up to the time of present trouble.

About May 1, 1911, patient was exposed to Neisserian infection; four days later the discharge from urethra appeared. He first noticed joint trouble in the right wrist. He states he did not injure his wrist. Pain, redness, and a little swelling and inability to move the wrist without causing intense pain were the chief symptoms. Wrist trouble cleared up in six weeks from onset. Three days after the onset of pain in the wrist the left elbow became involved. For eight weeks he kept the elbow at rest because of the pain, at the end of which time the elbow-joint was stiff. He had massage, passive motion, electric baths, and finally took gas to have the adhesions broken down. Arm was dressed in plaster cast and ankylosis recurred. Did not have a chill at any time.

COMMENTS AND OPERATION

DR. MURPHY: That is a fairly classic history. The patient denies having had a chill. The history does not state as to the stormy symptoms that preceded the metastatic deposit of the Neisserian microörganism in his elbow-joint. He had a mild urethritis, a specific urethritis, a purulent discharge manifesting itself four days after exposure to the infection. He went on for thirty days before he began to have pain in his wrist. That pain was not very severe or stormy; it was rather a mild manifestation of the metastatic joint infection, which is the exception. The elbow trouble began with a definite onset because he remembers accurately the number of days before he had pain in the elbow. His wrist recovered, which is common when you have arthritis as a metastatic manifestation of a Neisserian infection. His wrist got perfectly well and remained perfectly well. The inflammatory condition concentrated itself in the elbow-joint. This joint was immobilized during the acute stage of the lesion, and the result was a complete ankylosis. This was broken up under anesthesia and the arm put into a cast, again resulting in a bony ankylosis.

Let us follow the history and see where it deviates from the usual course. The rule is that in a metastatic arthritis from a urethritis metastases take place in the joint between the eighteenth and twenty-second day in 96 per cent. of the cases. He says that it was thirty days before he noticed the trouble. That is eight days after the usual time. That is not an accident. Frequently patients will say, "I got that because I was exposed in my automobile," "I got that because I got my feet wet," or "I got that because I was in a draft and had a chill," when the fact is that the chill is the manifestation of the metastasis. All types of arthritis are metastatic manifestations of infection in other parts of the body. I am speaking of acute arthritis. There is not a particle of doubt in my mind about that. It is an opinion based on a colossal experience, with careful analyses of the histories of the cases. The reason we have failed to find the primary lesion in the majority of cases with metastatic lesions is that the metastasis does not take place until after the primary lesion is forgotten. The patient was up and moving about when he got the "rheumatism." That is such a common expression, "Then I got the rheumatism." I recall the time very well when we had general sepsis in our hospitals, and with the metastatic joint inflammation came on pyemic symptoms. Exactly the same relations exist between arthritis and inflammation and infection of the mucous membranes and mucous cavities, like the gall-bladder, nose, throat, and tonsils, except that the metastatic infection is of the mild type and does not produce

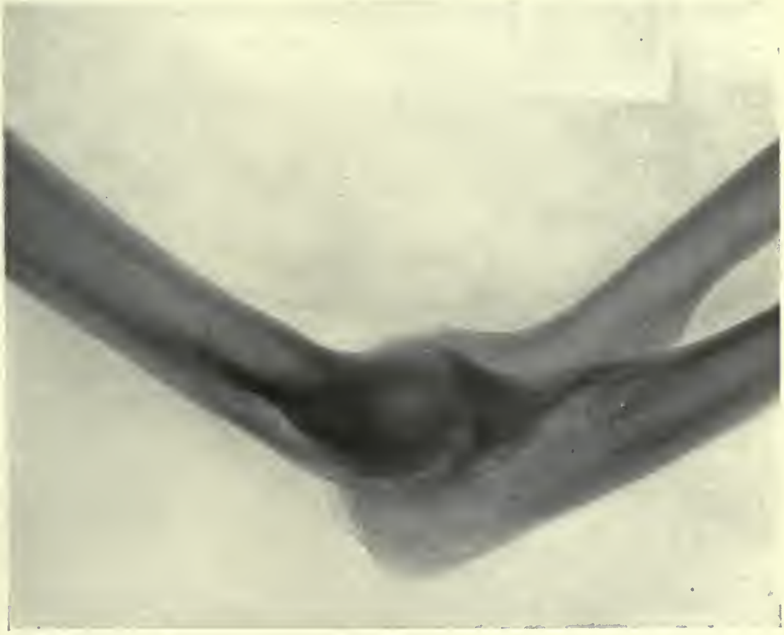

Fig. 133.—Complete bony ankylosis of radius to ulna and radius and ulna to humerus, with obliteration of joint anatomy.

pus in the joints. This is so significant, and so positive is my conviction on that score, that I mention the fact at every clinic, because on that basis every one of these cases is primarily a surgical case.

A joint lesion is a metastatic infection, and the final result depends on the virulence of the infecting microörganism and the management of that joint lesion. If these cases are handled properly, you can avoid all the frightful deformities so often seen by placing the limb in good position and maintaining that position during the inflammatory process. If you let the limb get out of position in the first few days, you will never get it straight. If you put the limb in good position with extension immediately, you will not only relieve the pain but prevent deformity and in all probability avoid an ankylosis.

Another matter that led to our undoing in this class of cases was aspirating these joints. We did not find any infectious material in the joint, and we therefore concluded that it could not be metastases. You may remember the extended and heated discussion we had some years ago as to whether certain joint infections were gonorrheal or not. One observer, say, examined one hundred cases and found gonococci in seven. That was looked on as a coincidence. Another man would examine the cases in another stage, and in 90 per cent. he found the Neisserian microörganism. It all depended on the time or stage of the affection that the examination was made as to whether or not any cocci were found. But it was always a secondary lesion, a metastatic lesion, with a definite, unvarying clinical course.

There is not a particle of motion in this elbow-joint. I will make the skin incision on the back of the arm, slightly to the inner side of the midline of the olecranon—an incision 5½ inches long, extending down to the bone, so as to give me free access to the joint. I am figuring for my interposing flap. You have to be governed by the amount of fat deposited. I am leaving all the fat on the aponeurosis of the muscle. I will take off the aponeurosis of the triceps muscle and interpose it later between the olecranon and the humerus when I separate these bones.

I will also use a portion of the capsule of this joint for that purpose. The aponeurosis is rather a dense fibrous mass. It is the type of fascia one occasionally finds between the ends of fractures and results in a failure of union. That knowledge led me to do this arthroplasty operation originally.

I will take the aponeurosis with the fat. The preparation of this interposing flap is the most important part of the procedure. I have made this external incision long enough so that it exposes the triceps. The fibers of the triceps and of the supinator longus, as well as the attachment of the long flexors of the fingers, are plainly visible. The olecranon, the tendon of the triceps, the external condyle of the humerus, are landmarks which are now in view.

The ulnar nerve is the one nerve that is likely to be injured in this procedure, therefore we will expose it to view by making another incision on the inner side of the elbow, parallel to the incision on the outer side. I do not wish to handle or compress the nerve during any stage of the operation, because in that way I could seriously interfere with immediate transmission of impulses by traumatizing its axons. With the nerve standing out very clearly and distinctly, we can keep it out of harm's way. I can now separate the radius and ulna and humerus from each other without in any way hazarding the ulnar nerve. It will not be interfered with at all.

Now we are going to separate the bony union. There is an ankylosis in this case between the ulna and the radius, as well as between the radius and the humerus. There is not a particle of motion. The line of union between the humerus and the ulna is easily located. The head of the radius is difficult to make out. Having definitely located the neck of the radius, I will remove the head of the bone, taking with it the periosteum, as if we leave the periosteum in place here it will be a great detriment to the restoration of motion subsequently. I chisel the radius free from the ulna, and then remove the head of the bone, about one inch, with bone-cutting forceps. That separates these two bones well, and leaves things at about their normal conformation.

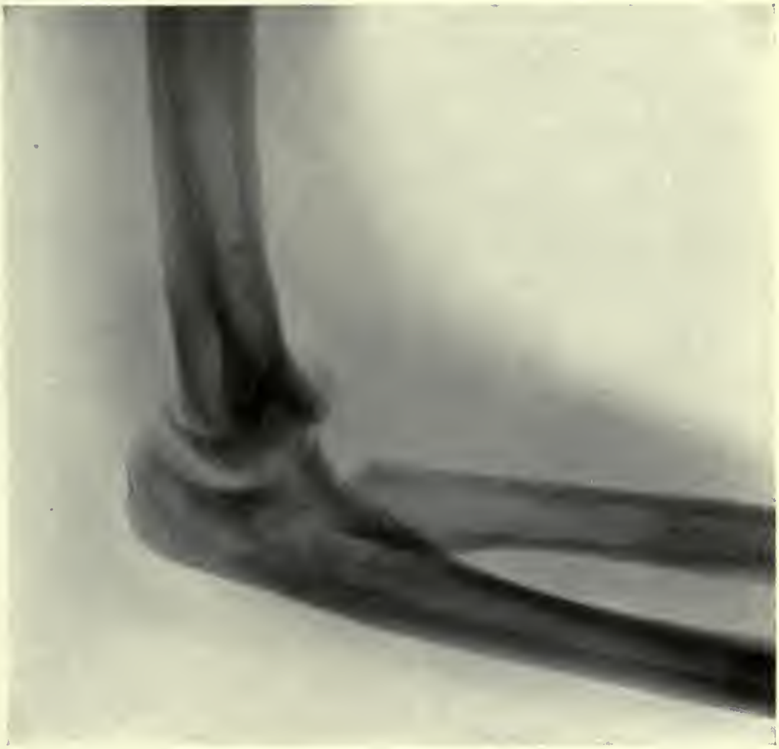

Fig. 134.—Skiagram taken four weeks after operation. Head of radius was removed. The cleft between the olecranon and the humerus shows that the interposing fat and fascia flap fulfilled its function. The motion in the joint was returning rapidly.

Now we must get the ulna freed from the humerus, which is not at all so easy a task. Now you see the advantage in having that ulnar nerve out of harm's way. You can see how well I have protected that; I can cut in here between the coronoid process of the ulna and the humerus in order to make my bone division without hazarding this nerve in the least. We will carefully chisel free the olecranon from the humerus, so that we can look right through the joint from one side to the other.

Now we have the bone free without having fractured it. We next slowly stretch the soft parts. Having removed the head of the radius, we have reëstablished pronation and supination. Pronation and supination are very important matters in these cases. The next step is to remove enough of the coronoid process and of the humerus to permit the interposition of the fat and fascia flap.

With chisel and forceps we have changed the conformation of the lower end of the humerus to about normal. I will be ready for my interposing flap after I have taken off the necessary amount of the coronoid process in front. That is one of the greatest annoyances in this work. That coronoid process must be removed, separating the tissues in front gently and leaving the periosteum on the bone. I shall remove the periosteum of the coronoid process, otherwise it will reproduce bone there, and that is a very great hindrance in the subsequent course of the case. You can see how easily I have separated the coronoid process from all its structures in front all the way across. You have a full view of the conformation of the new joint.

I did not prepare this flap until I had all my work done, to see just what style of flap I would need. I take this flap from the aponeurosis of the triceps, base directed upward, and the flap long enough so that it will swing into position between the olecranon, articulating surface of ulna, and internal condyle of humerus. I will pull this flap between these bones and suture it in place with catgut. A similar flap is brought in from the outer side, covering the external condyle. There is perfect interposition between these bones. We throw the ulnar nerve back into position; we are all the time keeping the ulnar nerve out of harm's way and protecting it. See how nicely that goes back in place, not contacting with the bone, but with the internal fat and fascia flap which I placed under the nerve for the special purpose of preventing a recurrence of the ankylosis, and for the second purpose of taking care of that ulnar nerve.

When you look at this elbow now it does not seem as though we had done much. It looks as though we spent a lot of time doing very little work, and when you get the stitches placed there is little to show for the work.

One of the greatest dangers in all this work is the danger of infection, as we handle so many instruments and do so much manipulating of the joint structures. We have been fortunate—I say fortunate, because no one has a supreme confidence in his asepsis except those who have had little experience. Up to date we have been fortunate in these cases in not having much infection. We had one man with sepsis of the hip; it was the second hip arthroplasty done for a double ankylosis of the hip. He got the infection on the tenth or eleventh day. He had been a morphin victim. He was not getting enough morphin, so one day he tore his dressings loose in the night and scratched the wound with his cane. He got an infection and died three weeks later. This is the only death we have had. We have had mild degrees of sepsis in other cases.

There is some oozing of blood, but I think it is only a venous oozing, and with the ordinary dressing pressure all that will subside. We rarely ever have to put on a ligature to stop hemorrhage in joint work. There is always some oozing from the surface of the bone that was divided.

This gives us now full and complete rotation, pronation, and supination. We divided the attachment of the ulna, its olecranon and coronoid process, from the humerus, which was the bony ankylosis. This was done with the curved and straight chisel and was not difficult. When it was free, a considerable portion of the tip of the olecranon was removed and a portion of the coronoid process well down toward its base. Then the intercondyloid groove and the trochlea were enlarged, and a portion of the end of the humerus removed, including both the condyles and the intercondyloid structures. The internal flap was interposed between the internal condyle and the olecranon and the base of the triceps tendon. The external flap covers the external condyle, passed between the radius and the humerus on the end and between the radius and the ulna on the side.

The technic of this operation varies from our original technic in this: In the original technic we divided the olecranon from the shaft of the ulna. Now we do not do that. We leave the olecranon intact all the time. It has this advantage: that early passive motion can be instituted, and that is a very important factor. We have not had, since we have followed this plan, any sloughing of the skin-flap over the olecranon and the passive motion will not hazard that interposing flap at all, will not put the line of union materially on the stretch, so we can start passive motion very early and get a more early movement.

The next and last step is to suture the skin incision with horsehair. I am forcing out the blood that is just beneath the skin so as to get it empty before we put on our compress. We want as little blood in there as we can possibly have, and now is the time to force out that blood. That acts not only as a sealing device, but it mobilizes or splints the skin, helps to keep it steady, and supports the line of union. We dust the wound well with bismuth subiodid powder and seal it with collodion gauze. This is the carbolated gauze you have heard me speak of before. That carbolated gauze has the advantage, as you know, of caring for the flap in an antiseptic way. It is placed over the collodion gauze. That dressing is quite loosely applied. We have no constricting dressings placed on here. We dress that arm at just a little less than at a right angle, so as not to compress the interposing flaps. A plaster-of-Paris cast is then applied, and when it has set we split the cast on the lower side with a Gigli saw, to give some play, if there is any swelling of the arm subsequently. We want that arm dressed in the elevated position. All that you see is done with a definite purpose from the very beginning to the end of the operation, to get a proper interposition. The horsehair stitches will be removed in ten days or two weeks, and then passive motion will be begun. In three or four weeks the patient will be encouraged to bend the arm himself, so as to restore the normal function of the joint as soon as possible.

ANKYLOSIS OF RIGHT HIP-JOINT

HISTORY

SALESMAN, age twenty-one. Entered hospital because of ankylosis of right hip and limitation of movement in right knee-joint. Family and personal history negative up to time of present trouble. March 2, 1911, patient exposed himself to a venereal infection. March 7th, five days later, he noticed a creamy yellowish discharge from the urethra. After treatment for two weeks the discharge ceased. April 11, 1911, just nineteen days after the disappearance of the urethral discharge, he experienced slight pains in inner aspect of right thigh, six inches above the knee, not severe enough, however, to prevent his keeping social engagements on each of the three following nights, the last of which was for a dance. He distinctly recalls having become quite warm while dancing, and perspired enough to dampen his clothing. After the dance, at one in the morning, he drove two miles in an open automobile. There was snow on the ground, and it was quite cold; he became chilled. On reaching home, before he could get half undressed, an intense pain seized him in the right thigh, just where previous pains had been felt. He said it came without warning, and he was forced to lean against the bookcase for support. It persisted without abatement, and he hobbled over to the bed and had great difficulty in moving his limbs enough to get in under the bed-covers.

He is sure he had no chill and did not vomit, and thinks there was but little fever. The hip was very tender that day, and this persisted for weeks. It was not swollen, red, or painful, nor has it been since that time. As he lay in bed he never turned on that side. He was taken to the hospital the day following the dance, and a Buck's extension with a five-pound weight was applied to the leg. He protested vigorously against this, and says he got the most ease by having the leg and hip partly flexed, with the thigh adducted, slightly throwing it over the other limb. He lay turned away from the diseased side with his weight mostly on the opposite side.

Two days later the extension was removed and a plaster cast was applied to the leg, extending from the axilla to the ankle. It was very tight and was placed entirely about the body and the right leg. It was not cut through to relieve tension, and caused him much pain. He wore this cast for fully five weeks, and large pressure sores formed on the heel and the back in the lumbar region, the latter extending for at least 6 inches directly over the spinous processes of the vertebræ. He lost 50 pounds in weight.

Seven weeks after onset of pain in thigh the tenderness in hip-joint still persisted. A doctor said the cavity of the hip-joint contained pus and advised an operation. The hip was laid open by an incision 5 inches long, and a large rubber drainage-tube was inserted, but the pus failed to appear. The tube remained *in situ* and the wound was open for two weeks.

Following this drainage operation the joint became completely ankylosed. In August, 1911, the physician attempted forcibly to flex the thigh under anesthesia. The flexion occurred all right, but the point of motion occurred below the joint, in the shaft of the femur, and today that limb shows over one inch of shortening.

Right knee cannot be flexed to more than an angle of 30 degrees. Right hip-joint is completely ankylosed. Upon examination of the heart a systolic blow was heard over the third left interspace. Apex-beat within nipple-line. Pulse irregular, rate averaging 66. Temperature normal; urine normal.

COMMENT AND OPERATION

DR. MURPHY: The discharge ceased nineteen days from the day he exposed himself.

THE INTERN: Two weeks, I think.

DR. MURPHY: No; the discharge ceased in nineteen days. The last Thursday in March, 1911, this patient exposed himself to venereal infection, and the Tuesday following, five days later, he noticed a creamy yellow discharge from the urethra. After treatment by a doctor the discharge ceased. He was not treated before he noticed a discharge. He was not treated until he noticed it, so that the discharge ceased at the end of nineteen days. I will show you in a moment that every day counts and must be counted.

THE INTERN: Tuesday, April 11th, nineteen days after the appearance of the discharge, patient experienced a slight pain on the inner aspect of the right thigh.

DR. MURPHY: He was sure he had no chill after the pain began, but he was chilled and felt cold in going home from a dance.

THE INTERN: He did not have a chill, but he felt chilly and cold.

DR. MURPHY: What is the story now?

THE INTERN: A drain was put in.

DR. MURPHY: Did you insert the drain?

THE INTERN: I was told about a drain being inserted.

DR. MURPHY: The drain was kept in how long?

THE INTERN: It was kept in two weeks.

DR. MURPHY: And following the removal of the drain, what was his condition? He must tell the story. The examination is what we made afterward. What was the sequence of the disease? What was the sequence of the drainage? What was the sequence of the pressure? And why did he come here to be examined? That is what we want to know. He is here to be examined. He is unable to get motion in his hip-joint. The record does not show that that hip-joint is ankylosed.

THE INTERN: The examination does.

DR. MURPHY: That is not it. That is our opinion of it. That is not the history. The history is that from the time the drain was removed the joint was ankylosed and has been stiff. There was some effort made, as I understand, to get motion in the joint, according to the first record.

The history is a record of the patient's statements. I want you to have the record right, and the way to make a diagnosis is on the record, and on the record as given by the patient, particularly the order of facts. Now, we find out how nicely the order of facts fits right into the pathologic condition, and why this man is here.

The Intern: In August, 1911, the thigh was flexed under anesthesia.

Dr. Murphy: The point of motion occurred below the hip-joint; he felt something snap when it was done, and the doctor believed he had a fracture. The skiagram does not show a deformity as the sequence of a fracture, but it shows there is some deformity resulting from the break. What date was that?

The Intern: August, 1911.

Dr. Murphy: That was a fracture of the neck. We can fracture it in the neck, throwing it out now, notwithstanding so long a time has elapsed since the injury. The story is what? It is a classic story. The dates are exactly right. He had a primary Neisserian exposure, and with the Neisserian exposure infection commenced immediately. Nineteen days after his exposure he had treatment in the regular way, but he got an additional exposure. He charges it to the ride home in an automobile and to exercise. Possibly that was it. I don't believe it, because he had pain in the hip preceding that ride, and he informed us that he was out in the rain that day, and the next day the joint was swollen. There was a second exposure, causing the metastasis to localize itself at one particular point or in multiple points in his joints. Here the metastasis took place in one joint. There is a peculiarity about the exposure when it attacks only one joint. No matter whether it is a Neisserian infection, tonsillitis, bronchitis, pharyngitis, or whatever the primary lesion may be, when the metastasis only attacks one joint, the attack is much more severe and much more likely to lead finally to bony ankylosis. One of the things that should be suspected immediately when only one joint is attacked is that it is going to result in an ankylosis.

Now, let us take up the management of the case.

Shortly after the manifestation of the joint trouble a Buck's extension was put on, but that did not give him relief, and the reason why it did not give relief was because the weight was not heavy enough. Five pounds is too little. You might as well put on a postage-stamp. Buck's extension is put on for a purpose. It is put on with cortical cells, and not merely with weight and pulley and plaster. It is put on for the purpose of separating these two inflamed surfaces, and the weight must be sufficient to produce that separation. The weight must be enough to afford the relief that is necessary.

He was relieved after enough Buck's extension was applied. As far as Buck's extension is concerned, the treatment was right in principle, but wrong in execution. If they had put on 25 or 30 pounds, the man would have gone to sleep more readily than he would have done with a dose of morphin. He has an infection of the joint of Neisserian origin. Neisserian infection in 95 per cent. of all the cases takes place in the joint between the eighteenth and twenty-second day after the primary invasion of the urethra or mucous surface, and it always takes place at that time if the patient has a secondary extension of the inflammation in his urethra, as from sounding, or additional exposure or trauma. All these cases of so-called "rheumatism" are instances of metastatic infections. There is no such disease as idiopathic "rheumatism," any more than there is idiopathic peritonitis. They are all metastatic conditions. Here it is easy to trace the primary lesion, because his discharge from the urethra continues. Metastasis never takes place in streptococcic infection except after the primary infection. It takes place in from twenty to thirty days, depending on the virulence of the infection rather than on the resistance of the patient, if it takes place at all. That is why for so many years we failed to connect the primary infection with the metastatic infection. It comes after a long span of time has elapsed. In the urethra we can establish the connection because of the discharge continuing, but there must be a definite pathologic reason for the metastasis taking place between the eighteenth and twenty-second day. There is a definite law governing this just as there is a reason for the sun appearing every day.

At the last clinic we had a beautiful illustration of an osteomyelitic lesion resulting from furunculosis of the arm, showing the time the furunculosis was present. It was fourteen days before the patient had her chill and a metastatic inflammation in her tibia, which caused the final necrosis.

In the management of this case, what happened? The joint was immobilized under great pressure. Wherever you have two serous surfaces inflamed, you must relieve the pressure. The synovial membrane is destroyed by two particular factors in infection of the joints. First, by the virulence of the microörganism that produces the synovitis; second, by the intra-articular pressure, by the involuntary contraction of the muscles forcing these two inflamed surfaces together. When we have an infection of a synovial membrane, if we would maintain separation of the inflamed surfaces; if we would avoid synechiæ, we must keep these surfaces apart. Here the surfaces were pressed together and the patient had pain all the time. He had not only pain from pressure in the joint, but he had pain from pressure of the mechanical support placed around his body.

We come to another proposition, namely, when you have infection of a serous surface, how are you going to maintain the integrity of that surface? *By reducing the virulence of the infection at the earliest possible moment, by relieving the pressure of the products of infection, and by keeping that surface away from the external air*. That is a law. If you expose any serous surface to the air for any considerable length of time, the endothelial cells lining that serous surface are *always* destroyed and synechiæ result. Once you decide on opening and draining the joint and maintaining drainage for any considerable time, the best result you can get, is what? An ankylosis. The worst result you can get, if you let it alone, is ankylosis.

Remember, these cases are impersonal, and what I say does not apply any more to this case than it does to others. My remarks are all impersonal; they do not apply to this particular case or to that particular case, but to the group of cases. One gentleman who attended my clinic went out and said that I criticized a certain man's work. Never! I criticized the principle involved. That is all I talk about. A man does his best with the knowledge of his time and the period in which he was educated.

That is all he is expected to do, and any criticism I may make is made on that basis and in that spirit.

What happened subsequently? After this joint had been immobilized it was drained, the discharge came out, and finally the tube was removed, and here comes the regular sequence, complete bony ankylosis, one of the greatest misfortunes that can occur to a joint. This man has complete union of the head of his femur with the acetabulum. This is the sequence of, first, infection and, second, drainage.

We will now proceed with the operation. We will do an arthroplasty in the usual manner. A U-shaped incision surrounds the great trochanter, the base of the flap being upward. I take out with this flap, the fascia lata, from which I will make a lining for this joint. This U-shaped flap is about three inches wide and five inches long. Formerly I made this flap longer, but I found that the flap occasionally died as a result of that, and so I have ceased to make it so long, but I make the deep flap long. The fascia lata and fat are dissected from the under surface of the flap, remaining attached at the upper end. This tissue flap will be turned in to cover the head of the femur and be interposed between it and the acetabulum, so as to prevent a recurrence of the ankylosis. We have taken off a double flap. It is a skin-flap, and an aponeurotic flap. The gluteus maximus is pushed out of the way, and here is the gluteus medius and there is the trochanter. We are going to saw off the trochanter with a chain saw and turn it with its muscular attachment upward out of the field of operation. I shall, if possible, try to get under the pyriformis muscle and the obturator muscular attachments. I leave all the muscles attached to the trochanter. That exposes the joint nicely. I will divide the capsule, open it up, and get ready for the excision. The capsule has become adherent around the neck of the femur. We will free it from its attachment. Now I am cutting the pyriformis muscle. The first time I cut this muscle I thought I had cut the sciatic nerve, as it is just like it, but not so far away. I am just leaving some of this tissue so that I can resuture the muscle.

Let the record show that I am freeing the capsule from the neck and turning it out; I may utilize this capsule subsequently. This is slow work, separating the capsule from its attachments to the side of the neck of the femur. That exposes the line of union between the femur and the acetabulum. This is a case of bony ankylosis of short duration. It is of Neisserian origin. It should not be of the dense osseous type by this time. It should fracture easier. It should follow somewhat the normal conformation of the joint with a little effort on my part. Softening has taken place on the anterior side of the head of the femur more than on the superior. With a curved Buck chisel I will chisel out the head of the femur so as to maintain its normal rotundity. I have a fairly good head, about the right size, and about the right conformation. It came out just about right. It is a little elongated; that we must shorten because we will have to put in our soft tissue flap. We have very few special instruments in doing this work. You need no special armamentarium to do this work. We must deepen the cavity from which this bone came out. For that purpose we use an instrument called a reamer. It is attached to a brace and the acetabulum is reamed out to the desired depth. An end mill, which fits into the reamer, restores the normal conformation of the head of the femur. That gives us a normal hip-joint—both anatomically and functionally. The next step is to place the interposing flap between the head of the femur and the acetabulum, to prevent the recurrence of the ankylosis. This interposing flap is taken off from the gluteus maximus with the fascia lata, which I prepared in the beginning, with a long tail so as to have it sufficiently long to go all around the acetabulum. It is larger than my skin-flap.

Visiting Physician: What about the danger of necrosis of the flap?

Dr. Murphy: I do not know whether that flap dies or what happens to it. I do know that no union follows. I do not think it necrotizes. It is a connective-tissue flap, as you see, taken from the fascia lata, which has feeble vascularity. It is sutured in place with a phosphor-bronze wire suture. I am suturing this to the capsule on the anterior side, to the capsule

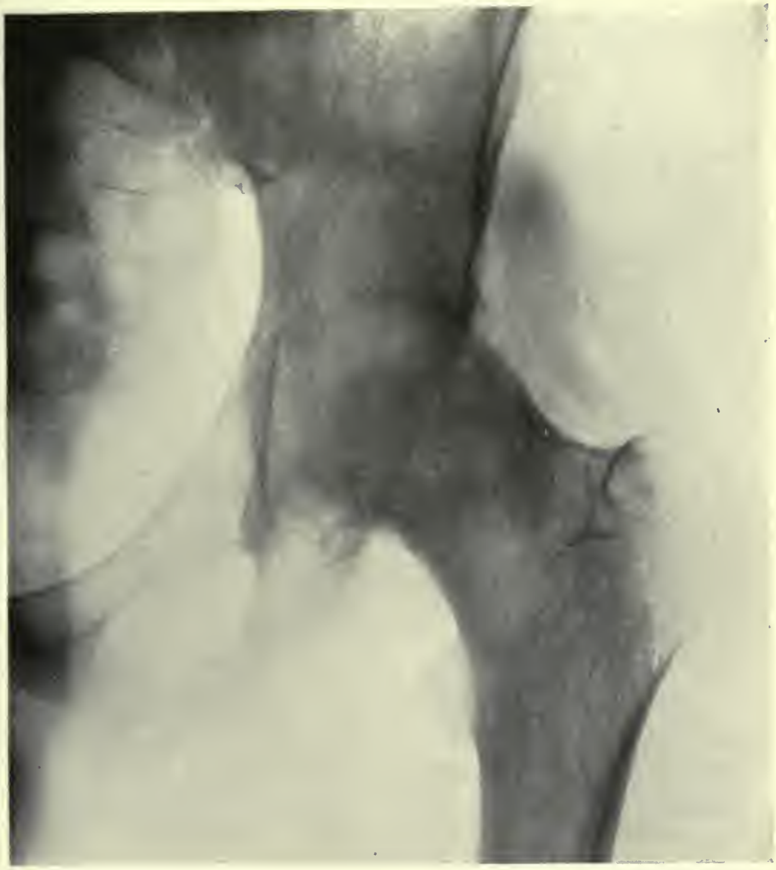

Fig. 135.—Skiagram taken before operation, showing complete bony ankylosis between head of femur and acetabulum with complete obliteration of all normal anatomy.

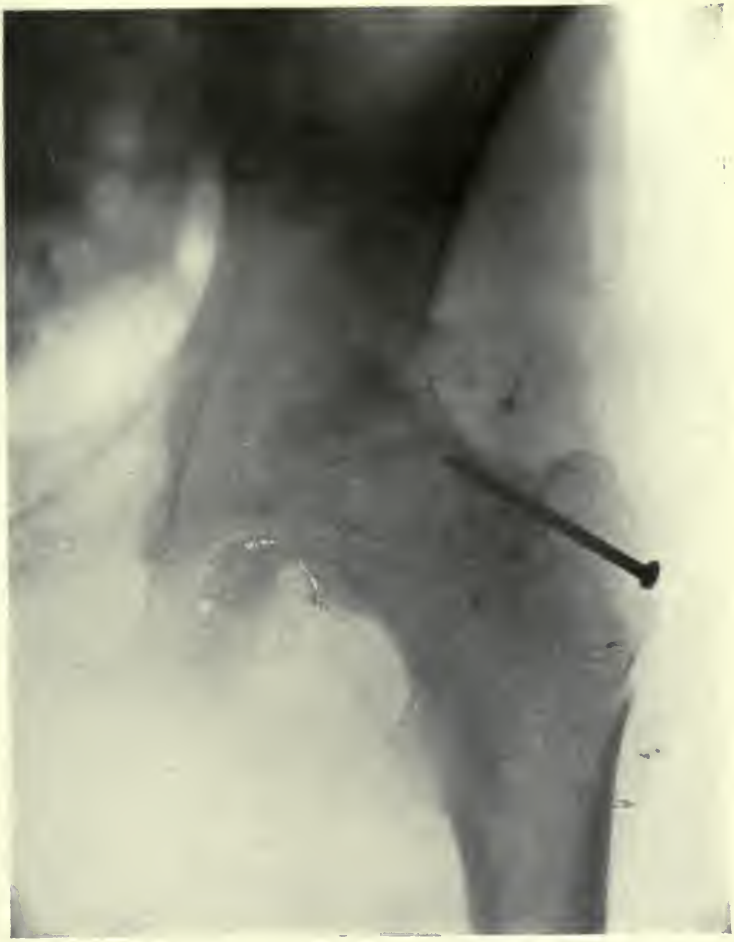

Fig. 136.—Skiagram taken immediately after operation, showing phosphor-bronze wire sutures in edges of interposed flap and wire nail transfixing trochanter.

on the posterior side, and to the capsule below. The bone will rest on that fat, muscle, and fascia, but principally the fat and fascia. You see how that is sutured to the margin of the acetabulum. These are just tacking stitches to keep all the raw bony surfaces covered. We learned in our pathology that if we interpose fascia or muscle between the two ends of the fragments of a fractured bone, you have failure of union. We have taken advantage of that pathologic knowledge by putting in these flaps, and it was on this basis that, ten years ago, we did our first arthroplasty. It was on this basis that we planned it, and this technic is identical with the original technic.

It was a hip-joint case on which I operated for the first time. We had occasion to demonstrate that case at Seattle before last summer. The patient was from Everett, Washington. I saw him nearly nine years after I did the first operation. He has a splendid anatomic result, with perfect motion in the hip. He rides horseback, and does anything that any young man of his age can do.

This flap is anchored all the way around to the capsule of the joint, so that it cannot become misplaced. You can put it on that way or suture it around the neck of the femur, which we have done, and turn it back. I like this plan—it is more precise and does not displace any of the restored fragments. We are ready for the restoration of the bone. We force all the blood out of this field. The head of the femur is easily placed back into the acetabulum by flexing the leg on the thigh and adducting it slightly. It stays perfectly in place. We suture the anterior portion of the joint capsule to what is known as the Y ligament, that caused so much discussion in the last decad of surgery. That immobilizes the bone. Every step of this operation we carried out on the cadaver before we attempted to do it on the human being. All our operations have been successful. The ankylosis has never recurred, and the normal anatomy and function of the joint have been restored. These patients never return for a second operation.

We have had many combinations of this operation that we do not count as true arthroplasties.

Now we are ready to do the next step, which is the fixation of the superior portion of the capsule to the acetabular margin with phosphor-bronze wire. That closes up the joint. The next thing is to restore the trochanteric group of muscles. These are restored by simply nailing the trochanter back in place with an ordinary wire nail. The next thing is to unite these muscles where I separated the fibers of the gluteus medius in front, and the anterior portion of the fibers of the gluteus maximus. You must not only have a movable joint, but a controllable one. That is why it is important to get the muscles back in accurate apposition. The bleeding is all stopped. We will not put on a ligature; we prefer not to use ligatures. That keeps the attachment all behind, so that we have good muscular apposition right from the beginning.

That completes the operation so far as the joint is concerned. The skin incision is closed with silkworm-gut and horsehair sutures. A collodion gauze dressing is applied after the wound has been dusted freely with bismuth.

The further treatment is to put the leg in an extension splint, with weight and pulley on to prevent compression and necrosis of the interposing flap. This patient is to have a twenty-five-pound weight attached to a Buck extension right away. The muscular aponeurosis was approximated with a few stitches, and that takes the place of the fascia lata, so that there is no danger of hernia on account of the muscle giving way.

The limb will be immobilized for about four weeks. We shall let him work his knee up and down, but we keep on the weight for two and a half weeks. After immobilization for four weeks we commence to put the limb through a little motion, and then we begin to lessen the traction weight when he is walking around. We put the traction on at night when he is in bed, to lessen the pressure in the joint.

Let me recapitulate the technic of the operation: First, make a U-shaped incision, with its base an inch above the tip of the trochanter major. Have the flap three inches wide and five inches long. That goes through the skin and about two-thirds of the superficial fatty tissue. An incision is then made parallel to the long axis of the thigh from the tip of the flap downward for a distance of three inches, so as to expose a larger area of the fascia lata than is exposed by the U-shaped incision. The fascia lata is then divided through to the subfascia lata, the loose areolar tissue, clear up to the level of the base of the flap. This flap is three-quarters of an inch wider than the U-shaped flap at its lower angle, and about its width at its upper. The skin flap and the fascia lata flap are carried upward and freed from the trochanteric bursa. The fibers of the gluteus medius are then divided in front, and the neck of the femur exposed just anterior to the trochanter major; the continuation of the fascial portion of the gluteus maximus is carried upward with the flap, and the gluteus maximus fascia divided so as to expose the posterior surface of the trochanter major. The fibers are then clearly outlined, the pyriformis and obturator tendons divided as they pass down through the digital fossa anterior to the trochanter, leaving them so that they can be subsequently sutured. A chain saw is then thrown around the trochanter with all its muscular attachments intact. The trochanter is sawed off parallel to the long axis of the cervix of the femur; the capsule is then loosened from its attachment to the neck all the way round; the quadriceps femoris is maintained; the upper fibers of the lower segment of the gluteus maximus are divided; the capsule is shelled from its attachment all around the neck, but left attached to the margin of the acetabulum. With a chisel the head is freed from its attachment to the acetabulum and is molded to the normal size and shape. We divide the adductor tendon so as to get the tension off. The leg comes out into position well and will be dressed in full extension and slight abduction with the Buck extension on. The interposed fascia lata flap was sutured all around the margin of the acetabulum and to the base of the capsule, so as completely to cover the circle of the acetabulum, and is pressed into it to make a pocket into which the head of the femur is inserted. This suturing is done with phosphor-bronze wire. The femur is drawn downward, the head rotated forward, and slipped into the acetabulum and onto the interposing flap. When it is in position, the anterior and posterior portions of the retained capsule are sutured again to the neck near the trochanter. The stumps of the obturator internus and pyriformis are reunited; the trochanter is then nailed to the femur in its normal position with a wire nail. The deep fibrous extension of the vastus externus is united with three phosphor-bronze wire sutures so as to give additional support to the margins. The separated margins of the fibers of the gluteus medius in front are then reunited, the same behind, so that the muscular connection is reëstablished on exactly the same basis that it was originally. The U-shaped skin-flap is closed with silkworm-gut and horsehair and accurately approximated. There is very little bleeding. No ligating need be done. No drain. The limb is dressed in the abducted position in a Rainey travois splint, with 25 pounds of Buck's extension applied. In putting on this travois splint you have every purpose accomplished. We have always endeavored to dress limbs in the abducted position, with the result that when we thought we had dressed the limb in an abducted position it was really an adducted position, and that is the reason for putting the limb in a Rainey splint. This splint is adjusted so that you can spread it any desired distance by loosening two bolts. It is a simple affair, and every practitioner should have it. (See Figs. 23 and 24, p. 173, vol. i, No. 2.)

The essential part in treating fractures of the neck of the femur is to keep up abduction and extension. If there is no interposition of the fibrous capsule between the fragments you will get union. If there is interposition of the fibrous capsule between the fragments there will be non-union; that is, when a fracture takes place, if the capsule is torn and, when the fragments are reduced, the capsule is pushed between the fragments, as was the condition in four consecutive cases we had here at one time, you will get no union, no matter what dressing you put on. We operated on these four cases and demonstrated our findings at the operations. We took out the capsule, put the fragments in apposition, and got perfect union in all the cases.

Let me say a few words in regard to the management of this case of Neisserian infection from its inception. First, every case of acute arthritis is a metastatic infection, and a careful analysis in a very large percentage of the cases will enable you to locate the primary infection atrium. It does not matter whether you are able to locate it or not, if the acute arthritis involves one or two or three joints, and has associated with it a chill, the clinical diagnosis or rather prognosis is that one or two or may be more joints are to be ankylosed. Why are they going to be ankylosed? First, because of the virulence of the infection; second, from the fact that muscular contraction presses these inflamed surfaces into each other and destroys the synovial lining, and finally, the cartilage, and, third, the products of infection are held in the joint under great pressure. How do you avoid destruction of tissue from infective products in other positions in the body? If you have a phlegmon of the thigh, a phlegmon of the leg, or an osteomyelitis, what do you do? Open it, to reduce the tension caused by the products of infection. That is all you need to do. Nature cures it. The same principle applies to joints, with the exception that if you open a joint and permit the air to enter, you are going to destroy the synovial membrane. But the same principle must be carried out in relieving the tension from the products of infection in the joint and relieving the virulence by the same means, except not through a large opening, and not with the insertion of a tube.

The first thing to do when you are called to see a case of acute arthritis, whether you call it "rheumatism" or metastatic arthritis, is to relieve the intra-articular pressure and the muscular contraction, and that is done by putting on a Buck's extension. In the small joints there is but very little pressure from muscular contraction, and therefore little tendency to ankylosis. If you fracture small bones, you do not have to put on extension, as they stay in position without extension. In fractures of large bones you do put on extension. That is number one. Number two, you relieve the tension from the products of infection in the joint. This you can do by aspiration, inserting a large needle into the joint, and, without irritating or traumatizing the synovial lined surfaces, draw off the fluid. The joint cavity is a connective-tissue bottle that stands an intra-articular pressure, as demonstrated by Dr. Kellogg Speed, of from 40 to

120 pounds before it ruptures the capsule of the knee-joint and of the hip-joint. It has a non-vascular lining, which does not readily destroy the infection. That is why it holds. In other places the infection breaks through, but not at the knee or the hip, because of the leathery capsule on the outside that is non-vascular and difficult to infect.

As I have said, relieve the pressure by aspiration; render the surface and joint a bad culture-medium by injecting into the joint a fluid that stimulates polymorphonuclear leukocyte protection. An ideal remedy for that purpose is a 2 per cent. of formalin in glycerin mixture. There is a preparation of mercury, but it is too toxic to be used. Use 2 per cent. of formalin in glycerin, mixed twenty-four hours before it is injected. If you inject it into the vein of a dog, it acts as a cardiac stimulant. If you inject it into the synovial membrane, it acts, first, as a stimulator of the polymorphonuclear leukocytes, which is an important factor; second, it is a medium in which the microorganisms will not grow, and, third, it stimulates infiltration and coffer-damming of the synovial membrane and stops absorption. One or two or three aspirations of a joint, such as we saw today, in the early stage, will avoid the necessity for opening the joint. It stops infection, and the case goes on, as we have shown here in this clinic, without the slightest tendency to ankylosis of the joint. It stops the whole thing as a primary lesion. The treatment is the same in all cases, no matter whether the primary infection is grip, pneumonia, or typhoid. They are all alike. They are all metastatic inflammations, but the clinical fact remains that the less the number of joints involved, the greater the destruction of the joints.

VOLUME I *NUMBER 6*

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OF
JOHN B. MURPHY, M.D.
AT
MERCY HOSPITAL, CHICAGO

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NOTE: In the February, 1913, number of the Clinics will be published an address by W. Arbuthnot Lane, of London, delivered at Dr. Murphy's Clinic November 23, 1912, on the subject of the Treatment of Fractures of the Long Bones With Plates, in which he explains the causes of so many failures in this treatment and gives a very clear description of the technic. Mr. Lane plated a fracture of the tibia, doing the operation before the assembled doctors, and Dr. Murphy gave a very clear exposition of Mr. Lane's method of operating.

All this will appear in the February, 1913, number.

SURGICAL CLINICS
OF
Volume I JOHN B. MURPHY Number 6

CARCINOMA OF BREAST

WITH A TALK BY PROFESSOR R. BASTIANELLI, OF ROME, ITALY

HISTORY

PATIENT, a married woman, forty-nine years old, came to Mercy Hospital, August 24, 1912, on account of a small tumor in her right breast. On Sunday, June 30, 1912, while taking a bath, she accidentally discovered a small nodule in her right breast. It was about the size of a small walnut. She does not think that it has grown any larger since then, but says she can feel it more distinctly. She gives no history of an injury other than that her husband accidentally struck her on that breast with his elbow "three months ago." She never had any discharge from or eczematous condition of the nipple. Has always been healthy. Menstruation regular. Her first pregnancy terminated in a miscarriage at the seventh month. Has two children, the youngest fourteen years of age. No history of tuberculosis or malignancy.

Examination reveals a hard nodule, about the size of a walnut, near the periphery of the right breast, at the junction of the upper and outer with the lower and outer quadrant. It is neither adherent to the skin nor to the underlying structures. Circumscribed, but not very definite in outline. It does not disappear, but becomes more distinct under pressure with the flat of the hand. The remainder of the breast is normal. An enlarged gland, hard and movable, is felt beneath the pectoralis major muscle at the margin of the anterior axillary fold.

COMMENTS AND OPERATION

DR. MURPHY: This is a fairly typical history, except for the trauma relation. The relation of trauma to a carcinoma of the breast is a very peculiar one. It differs from the trauma relations to carcinoma in other positions of the body. While we have learned little of the true etiology of carcinoma, we are fairly uniform in our belief that carcinoma is the sequence of repeated mild irritations—not of one severe irritation, but of repeated mild irritations, whereas sarcoma is the sequence of one moderately severe irritation or trauma, never a superlative trauma.

Let me make that clear. Carcinoma of the lip does not occur as the result of striking the lip with the fist and splitting it or cutting it, but it occurs on account of a continuous mild irritation from a tooth. A sarcoma never occurs in a bone following injury where the trauma is severe enough to fracture that bone. It does occur, however, where the trauma is severe enough to cause pain, like striking the tibia, for instance, with a baseball, with a bat, or striking it against the chair. When the trauma produces a fracture, there never follows a sarcoma. I say "never," because I do not know of a well-authenticated case of sarcoma originating in a fracture. There are fractures associated with sarcoma, but these cases have been pathologic fractures, where the sarcoma was present at the time the fracture occurred—in fact, it was the cause of the fracture.

In this particular of mild trauma sarcoma resembles tuberculosis. Tuberculosis of bones and joints is precipitated, in a large percentage of cases, by a trauma of mild severity, never one of superlative severity. For instance, a child has an injury to the knee, or falls and has an injury of the head of the tibia, and in two and a half or three weeks after the injury, after the incident is practically forgotten, the child has difficulty in walking, and finally the injury results in a true tuberculosis.

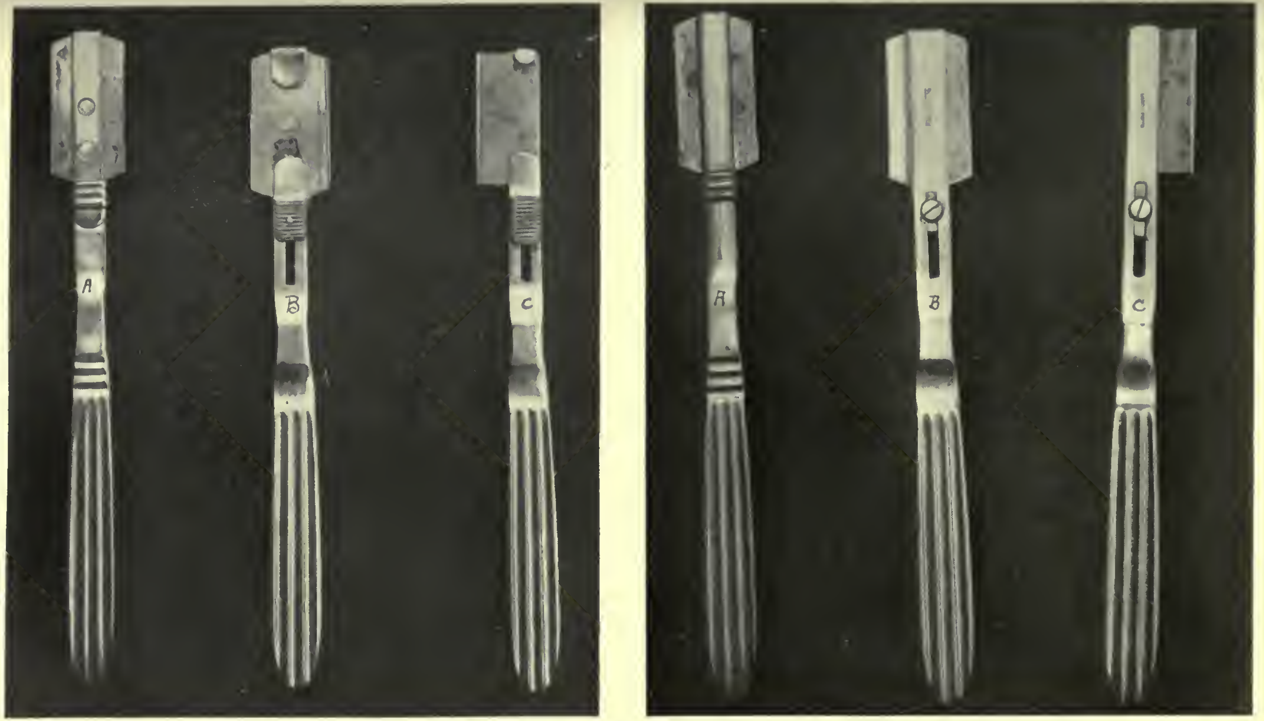
In the breast we have an exception to this rule, so far as repeated traumas are concerned in their etiologic relation to carcinoma. In the breast you may have one moderately severe trauma, and six, eight, ten, twenty, or thirty months afterward there is noticed a carcinoma. The breast is the one exception in which a *single* trauma of moderate severity produces a carcinoma. In traumas of the testicle a carcinoma occasionally occurs as the result of a moderately severe injury, but it is not at all frequent. Sarcoma occurs as the result of a moderately severe injury of the testis. Just what rôle trauma plays in the production of sarcoma we do not know. Whether it plays a rôle similar to that of the trauma in tuberculosis by the precipitation of some unknown microörganism, or whether it plays the rôle of perverting the growth of the cell and converting the cell into a parasite, we do not know; but the clinical fact remains that the trauma is associated with the disease and is an etiologic factor in its precipitation. So far as we are concerned, repeated mild irritations are the etiologic factors of the carcinomata in certain definite positions, as on the lip and in the lacerated cervix of the uterus; in the irritations that occur near the pylorus of the stomach, where there have been ulcers and scars, and where there is a recurrent retention in the intestine of its contents, as the sigmoid and colonic flexures. In other words, wherever there is recurrent irritation, that is the site of carcinoma. Whereever you have intestinal stasis, that is, normal, physiologic, or anatomic stasis, you have what? You have the frequent site of carcinoma, so that in these cases we must remember it is possible to do some prophylactic work in the way of avoiding the occurrence of carcinoma by removing some of the causes, such as continuous mild irritation.

With regard to tumors of the breast, they are so classic and described so frequently in the text-books that it is unnecessary for me to go into that phase of the question at this time.

The tumor in this case did not begin in the usual place in the breast. It is situated in the periphery of the breast. It is perfectly movable; it is in a woman, forty-nine years of age, who has a large amount of adipose tissue, and she is just the type of woman in whom carcinoma is most dangerous ultimately. I have used the expression "most dangerous ultimately," for in this type of woman, when a carcinoma is discovered by the patient herself, the rule is that the tumor has gone beyond the possibility of surgical eradication. In the case of a small, thin woman, or in a woman of sixty-two years of age or more, weighing, say, 125 or 128 pounds, who develops a scirrhus or a hard nodule in her breast, that woman's life expectancy is enormously greater than that of this woman, because wherever you find an induration, hard and ligneous, in any tissue, that induration means what? That induration does not mean irritation: that induration means a response of the connective-tissue cells or the defensive cells against the irritation of the invader of the tissue—be that parasite or infective microörganism. What else does that mean? It means that there is present a coffer-damming of the affected zone to prevent the tumor cells or infective organism from getting away into other positions of the body. The patient we have before us has little or no such coffer-damming. These tumors, as a rule, are very much softer than the tumors that occur in thin, emaciated, small women. The connective-tissue encapsulating response to invasion is much greater in the aged and thin woman than in a woman of this type.

The question arises, is this a carcinoma? If it is a carcinoma, what are we going to do? It is movable. It is apparently smooth on its surface. It has no sharp edge. Whenever you find a tumor like this in the breast, you always hope that it is a cystic formation or an adenoma, and not a carcinoma. When you find the nodules with sharp edges, just like the sharp, woody edge of this disease in some of the organs in the abdomen, you conclude that it is a carcinoma, no matter where it is situated. So we still have hope that this lesion is not a carcinoma. If it is not a carcinoma, we will merely remove the segment of the breast in which the tumor is situated. On the other hand, if it is a carcinoma, we will remove all the breast and axillary glands but not the pectoral muscles.

It has been my practice for years not to take out the pectoral muscles. If I excavate the axilla, I fill the axillary space with the pectoral muscle. That is based on the original report made by Bryant, of London, in Guy's Hospital, who had seen but one case of recurrent carcinoma in the pectoral muscle in an experience extending over forty years. That is one argument that has appealed to me in the way of clinical observation against the extensive operation of Halsted, which we did for many years and then abandoned. When the carcinoma went through the posterior mammary capsule and into the pectoral muscle, Bryant took out the pectoral muscle. So do we. We utilize the pectoral muscle to fill up the dead space in the axilla and to prevent contraction of the fascia, which would impede the return circulation in the



Back Views

Front Views

Fig. 137.—Dr. Murphy's safety razor blade scalpel.

The blades are detachable. Two types of blades can be used—single and double cutting edge. The double has one edge protected by a flange, so as to obviate the element of danger. A sliding ratchet fastens and releases the blade. *A* shows how knife is fitted into a slot and held by the metal buttons and the ratchet. This was the first pattern. There is no protecting flange on this knife. *B* is a later model, provided with a flange, and the blade-holding part has been modified by eliminating part of one holding arm. *C* same style of handle as *B*, without a flange, but with single cutting-edge blade. These handles were originally made for Dr. Murphy by Sharp & Smith, Instrument Makers, Chicago, Ill.

arm. Thus we prevent the edema which occurred in 31 per cent. of the cases operated on at the Massachusetts General Hospital, according to the statistics collected by Dr. Greenough. He found, after collecting all the cases that were operated on in the eastern states, that 31 per cent. of the patients operated on for carcinoma of the breast had edema and swelling of the arm and many of them to the degree of incapacity. It is one of the things to be avoided.

The next matter that comes up is the line of transmission from the breast into the various positions in the body of the malignant or carcinomatous cells. We know that we have four essential chains of lymphatics, or, by adding Handley's, we have five. There is, first, the major chain, the one that goes up into the axilla; second, the anatomic chain, drained from the zone of primary infection; third, the chain that passes into the chest at the second interspace to the close side of the sternum; fourth, the chain that passes into the sixth interspace in the anterior abdominal line, and, fifth, the chain of Handley, or the chain of lymphatics which passes down to the umbilicus. By the way, I have a patient operated on four years ago for carcinoma of the breast who has a recurrence in the umbilicus, proving the correctness of Handley's position, so far as local recurrence is concerned, but I am not at all satisfied that Handley is correct in his assumption that a large number of carcinomata of the liver occur as the result of metastasis through this chain of lymphatics. I do not believe that is true.

I am making my incision in such a manner that I can continue it and take out the breast, if necessary. I will keep the scar line out of the axilla. That is the first thing to remember. A scar line in the axilla is as inhibitory to subsequent perfect mobility as one in the fold of the elbow or one in the popliteal space. I will encircle the breast, commencing the incision to the inner side of the anterior axillary fold, and then, passing downward and encircling the entire breast, meeting the first portion of the incision outside of the axilla. I will then elevate the breast from the chest-wall. I am not near the enlargement in the breast at all. I will take out the aponeurosis of the pectoralis muscle, lifting it up with the breast. In removing the cysts of the breast removal from beneath is the most desirable. It is the easiest because the great majority of these cysts are situated near the posterior capsule of the breast rather than near the anterior. The neoplasm is now freed entirely from behind. It has the smooth contour to which I called your attention. Coming now to the under side of the breast, I find that there is a hard mass there, which is suspicious. I will take out a V-shaped piece of this tissue, going wide from the neoplasm, and examine it macroscopically. I depend on that examination for my decision as to how radical an operation I will perform. One does not need a microscope to make a diagnosis of carcinoma in the majority of cases. The smooth round edge of the tumor which I felt before the operation is now apparent. The mass gives exactly the same sensation as that which is imparted by a cyst. Its incised surface shows that it is a carcinoma. Now we will remove the breast in the regular way.

Raising the breast and retracting it to one side, the aponeurosis of the pectoral muscle comes into view. I am going almost over to the margin of the sternum in removing this aponeurosis. That is a regular step in our procedure. The next step in the management of these cases is to get in between the pectoralis major and the pectoralis minor, and take out the glandular bearing zone of connective tissue that is situated there in the fascia between the pectoralis major and minor. I pass my finger in between the two muscles and detach the breast, lifting all the aponeurosis off of the pectoralis major with the removed portion of breast. Now we can go up into the axillary space and clean that out as we would if we excised the muscle. The pectoralis minor is separated from its costal attachment. We will clean out the axillary space from above. The pectoralis minor and the main pectoral vessel are in view. In this position you find the lymph-nodes, and not infrequently they contain carcinomatous deposits. I will remove the aponeurosis from the pectoralis minor entirely because, remember, the lymph-vessels do not pass through the muscles here, but pass on the muscles.

Next we push the pectoralis minor out of the field, and we are right in the axilla. We can palpate some enlarged lymph-nodes. I am in the upper portion of the axillary zone. There are the brachial plexus, the axillary vein, and the fatty tissue in which these nodes are situated. I start from the clavicle and denude the vein downward in place of following it upward, as we did formerly. I wish to get out not only all the lymph-nodes, but all the node-bearing tissue. I am wiping this tissue out with gauze clear back to the muscles in the posterior wall of the axilla. We are coming on to the nerves that pass down along the scapula and supply the latissimus dorsi. Here are both nerves in the field, as you can see from the faradic response I get. We preserve these nerves easily, going in from above downward. When you go from below upward, you have great difficulty in preserving them. We are commencing to separate the fat from the upper surface of the vein and pass away from the vein down into the axilla. We are away from the important structures of the axilla. There are the muscles on the posterior wall of the axillary space. You see, in wiping this space out I separated the lymphatic chain from above first, so that I am not in danger of wiping anything into the lymphatics. This is a short operation, when you consider the exposure which we have here and which you get always by going in from above downward. It makes it much easier than going up from below.

Here is the long subscapular nerve. Now, you see, we are entirely out of the zone of danger. I am taking this tissue off well back because this disease originated far in the outer margin of the breast. I want to get all the gland-bearing tissue out of that zone. There is a little resemblance to an angioma here, which I think has no relation to the tumor. It goes right into the skin, and I will remove it.

The next step is the fixation of the pectoralis major. Here is one of the glands removed from the pectoralis fascia, which appears to be the seat of a metastasis. A pressure occurs in the axillary vein with each inspiration. See it sink with expiration! That is one of the dangers of opening the vein in the axilla. It is with minus pressure that you can get air-embolism following.

If we permit that wound to heal without protecting the vein in some way, what must happen to the vein? The scar will cause a certain degree of compression of the vein, and that means a certain degree of stasis, and stasis means edema of the tissues of the forearm and arm. How can we avoid that compression? By two means: First, by filling in the lower space with the pectoralis minor. With one catgut stitch I can fix the end of the muscles, protect the major portion of the vein, and in cases where I have to remove the pectoralis major I use the pectoralis minor to cover that, but it does not fill the axillary space as well as when one uses both of these pectoral muscles. I close in the upper axillary space with one stitch, and another stitch closes the lower part. This muscle is sutured to the muscles forming the posterior wall of the axilla. Once that is filled with tissue it protects the patient against venous stasis, and, not less important, the compression of the nerves which causes the neuralgias which result from cicatricial contraction and, finally, we avoid fixation of the arm to the side by the contracting scar tissue. That is all there is to it.

What is the final prognosis? The prognosis, notwithstanding the fact that we have operated on this patient at an early date, is not very good. Why is the prognosis not good? Because a large gland removed from high up in the axilla is apparently involved. And she is a woman forty-nine years of age and very fat!

Are there other glands higher up in the cervical triangle involved? I do not know. Are there other structures involved, like the upper end of the femur, the body of a vertebra, the liver, or the brain—and I mention them in the order of frequency of metastasis? I do not know. I do know that in cases as simple as this we have had a patient taken with pain in the back before leaving the hospital, and subsequently sustaining a pathologic fracture of the spine. I do know that we have had patients with "rheumatism" in the hip before they left the hospital, because a metastasis occurred in the upper end of the femur. And still we are going on doing this operation. We are doing it because we believe that we are giving these patients comfort and that we are prolonging life. As regards the latter, I want to use a great big interrogation point. I am not sure that these patients would not live longer, as a total class, if they were not operated—not this class, because if I included this class I would not be operating on this woman. We will close the wound with silkworm-gut and horsehair sutures, approximating the skin-edges carefully and without tension. A small rubber tube drain will be placed in the lower angle of the wound. This drain will be removed in three or four days unless there is still discharge of fluid. A large dressing will then be applied with the arm well up on the chest. The stitches will be taken out about the tenth day.

I am pleased to inform you that Professor Bastianelli, of Rome, Italy, is with us today. He has had a very large experience with carcinoma of the breast, and, if he will permit me, I will quote him in regard to this class of patients. According to his own statistics, he thinks the final mortality approximates 100 per cent., and that confirms the position we have taken for so long a time.

I will ask Dr. Bastianelli to say a few words, and, if possible, give us a ray of hope in a field that is so dark and bleak as that of carcinoma. He has had an opportunity to observe in Rome some of the best work that has been done in the world along the line of therapeutics. He is a man of conservative judgment, based on a colossal experience and most careful observation.

DR. RAFFAELE BASTIANELLI, Rome, Italy: Professor Murphy puts me in a very difficult position because, in the first place, after having heard him speak with such authority on carcinoma of the breast one is naturally impressed, and in following him, embarrassed. Secondly, it is the work of many men throughout the world who are pursuing this line of investigation with regard to carcinoma on which I shall briefly report, and I feel I would like a little more time for reflection before talking about them: For this reason I will speak especially of the work of Professor Fichera, a young man in Rome who has devoted much of his life to the investigation of carcinoma. What is the origin of carcinoma? We do not know. There are two theories in regard to carcinoma that have been advanced. One is that it may be a parasitic disease, produced by germs as yet undiscovered;

the second theory is that carcinoma develops from epithelial cells which, by special conditions, are stimulated to abnormal growth by being perverted in their biologic state so that they penetrate in the connective tissue and continue to grow without limit. This second theory, from clinical or experimental investigation, seems to be more probable; but what are these special conditions or factors which push the epithelial cells on to new activity and make them grow indefinitely? You heard Professor Murphy make the statement that repeated irritations or repeated traumata, excepting in breast cases, are one of the causes of carcinoma which we clinically accept as effective. That is true. But we know also of many cancers starting without any external irritation. If you ask yourself the question, in what way a traumatic or chemical irritation acts, then you can easily answer, that these irritations are only an occasional cause—not the real and original one. Why does trauma or irritation contribute to make the epithelial cells become active? One theory has been advanced that through the traumatism the epithelial cells are detached from their connection with the mother tissue and are pushed into the connective tissue, and then, becoming independent, they grow. They can grow to a state of activity very much like embryonal activity. But why do the cells acquire this new property? There must be some factors inherent in the individual which tend to give to these cells new property or to subtract from the body something which normally regulates the growth of tissues. Professor Fichera has carried on a series of investigations for the purpose of studying how embryonal tissue grows when implanted into rats. If you implant embryonic tissue into rats, it grows for a while, but after a certain time the tissue is destroyed completely. There is no possibility of producing experimentally a tumor by injecting embryonic tissue into a living animal, but it is possible to make the grafts grow for a certain length of time or to make them grow less rapidly or to prevent or to stop their growth in changing some conditions of the experiment.

For instance: In animals previously grafted the new graftings are less and less apt to grow. It seems as though the animal had acquired an immunity against the embryonal tissue. This was proved not to be due to antibodies in the blood-serum. During the involution of the first grafting it was possible to see that the cells died through dissolution or autocytolysis, and it was thought that this process was providing the organism with substances capable of having an elective action on the cells of the new graft. In fact, preparing a liquid by leaving for a certain time aseptically embryonic tissue in salt solution, that is, in making an autolysis or dissolution of this tissue and then injecting it in rats successfully inoculated and having grafts in full growths, it was possible to see these grafts disappear completely. From that came the idea of trying the same in rats grafted with the so-called spontaneous cancer, which, biologically, runs in many ways similar to the embryonic tissue-grafting. The results were identical, and it was possible to stop and to cure animals having big growths which would have ended fatally, but not to immunize them against a tumor graft. The ulterior investigations of the factors which may favor or change the conditions of growth of embryonic tissue or tumor grafts have proved that these factors must be looked for in nutritive and formative substances produced by organs or glands of the body. Some of them favor the growth and they may be called *oncogenic*—that is, having the property of stimulating tissue to grow. Some act on the contrary—that, is, they stop or destroy the tumor,—and may be called *oncolytic*. These factors must be normally present in the body and regulate the normal growth, but occasionally the production of one or the other may be disturbed. It even may happen that a change in their mutual relations takes place, so that some cells or groups of cells already disturbed in their functions may be stimulated and pushed to an abnormal proliferation or not checked in their disordinate proliferation.

The embryonic cell is the one which more easily responds to biochemic actions. In our body are found groups of embryonic cells or tissues scattered in some organ. On the other side, it may be that groups of normal cells displaced by traumas or stimulated by repeated irritation are put in an abnormal reproductive condition which is very near to that of the embryonic stage. It is easy to think that these cells will feel more deeply the influence of any perturbation in the proportion between the oncogenic and oncolytic factors, and begin to grow without any possibility for the body to stop their progress. A close and systematic investigation of these organs and glands, which seem to have a greater influence on the growth of embryonic tissue and of experimental tumors, has demonstrated that the spleen and the genital glands act in opposite direction.

If you remove the spleen of an animal and then inoculate embryonic tissue, you see that the tumor grows much quicker, and in a greater percentage the graft takes place. If you remove the testicle instead, you will notice that there is either a complete check in the graft, or it does not grow as well as it grew formerly; and, on the other side, the injection of autolyzed spleen stops a prosperous growth, while that of an ovary makes it develop quicker.

From these investigations there have been therapeutic principles established. If you autolyze spleen or embryonic tissue and inject it into the system, you may give to the organism the autolytic property which it has lost. That has been found to be very effectively accomplished either by autolysis of embryonic tissue or by autolysis of spleen tissue. Applying these principles to rat and mice cancers,—experimental cancers,—they have proved most effective. I may say to the physicians present that we can check the growth of cancer in a mouse or in a rat by injection of the autolytic tissue of the spleen or of embryonic tissue, but as far as man is concerned I cannot tell you anything definite in the way of results. I know of some cases in which the results were favorable, and in one at least is satisfactory so far, but the time that has elapsed is too short, and it will require a great deal more experience before reaching a definite conclusion. This case was one of abdominal removal of the uterus and ovary for cancer in a patient in whom the disease was rather advanced. This is just an experiment in a woman to be compared to experiments in animals—that is, the removal of the tumor, plus the removal of the genital glands. In removing the genital glands you remove one of the oncogenetic factors of the tumor, and adding to the organism the autolytic property of embryonic or spleen tissue, you add the constitutional conditions or oncolytic factors that have been lost. This case was seen by myself when presented to the Royal Academy of Rome. The woman made a fair recovery from a rather extensive recurrence involving the vaginal scar and a part of the parametrium.

As to the outlook in the future of these investigations, Professor Fichera thinks that possibly some day we may find the autolytic properties of our organism which defends us against the invasion of tumors, and we may perhaps find the way to utilize them from a therapeutic point of view.

Dr. Murphy (resuming): I congratulate myself that I was so persistent in asking Professor Bastianelli to speak to you. He has given us a splendid, clean-cut statement of the situation, and a scientific analysis of this great problem, which must have impressed you greatly, and shows you that the reputation Professor Bastianelli has attained is not an accident, but a sequence. I thank him for what he has said. Two years ago I made a special trip to Rome to see Professor Bastianelli and his work. I knew that he was right in touch with this work on cancer, and if I felt it sufficiently important to make that special trip, you can readily realize why I was so persistent in asking him to speak to you.

[The microscopic examination of the tumor showed it to be a scirrhus carcinoma. The lymph-node removed, notwithstanding its enlargement and apparent infiltration, was free from metastasis. The subsequent progress of the case in the hospital was uneventful.—Ed.]

IMPROVEMENTS IN THE TREATMENT OF MALIGNANT TUMORS WITH RADIO-ACTIVE SUBSTANCES

By Albert Caan, M.D.
Heidelberg, Frankfurt a. Main

An Address Delivered at Dr. Murphy's Clinic, October 1, 1912

Dr. E. Wyllys Andrews: I have very fortunately the great pleasure of introducing to you this morning one of our distinguished German physicians. You are well aware that there are about 200 of the well-known members of the German medical profession who have been visiting us in the last two or three days. These gentlemen have been in attendance at the International Congress on Hygiene and Demography at Washington, and are now en route through America, visiting some of the principal universities. Fortunately, Dr. Murphy was able to persuade Professor Caan, of Heidelberg, who is connected with the Samariter Haus in Heidelberg, an institution where they have done very valuable original work on thorium and other radio-active substances, to tell you what they are doing. Gentlemen, I have the pleasure of introducing to you Prof. Caan, of Heidelberg. [Applause.]

Dr. Albert Caan: It is a great satisfaction to me to give you my experience in the treatment of malignant tumors with radio-active substances.

American science has done much to further the treatment of tumors with radium. Einhorn,* as early as 1904, successfully treated 7 cases of cancer of the esophagus with radium sounds. London,† in a patient suffering from cancer of the cecum, introduced, through the preternatural anus, a glass tube containing

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* New York Medical Record, 1904.

† Journal of the American Medical Association, 1904.

10 milligrams of bromid of radium, with the effect of reducing the tumor in size after one hundred and twenty hours' irradiation. Dr. Abbe* reported 11 cases of extensive inoperable giant-cell sarcoma cured by the influence of radium.

It would take too much time to mention all the authorities, American or foreign, who, with more or less success, have employed radium in the treatment of malignant tumors. I must, therefore, limit myself to the record of the improvements which have of late been made in the treatment of malignant tumors with radio-active substances. I mean to give an account, not of what has been done by celebrated radium therapists, such as Wickham, Degrais, Dominici, Exner, Finzi, and others, but of my own work as the assistant of Professor Czerny, at the Samariterhaus, in Heidelberg.

It is a known fact that the physicians I have named, as well as Professor Czerny, have succeeded in obtaining results with bromid of radium which one can almost describe as a cure for malignant tumors, and that in the case of superficial cancers (cancroids of the skin, epitheliomata of the lip, etc.) real cures have been effected; these facts have been acknowledged by known authorities. To attain these results it was necessary to have a sufficient quantity of radium to develop a special technic and empiricism, conditions which were fulfilled in the first instance by French radiotherapists.

I will also mention the metal actinium, which Debierne discovered in 1899 in the residue of protoxid of uranium, which Czerny and I have introduced as a cure for malignant tumors; hitherto its effects have been but evanescent, and it is nothing but a substitute for intratumoral injections of radium.

Of late much has been written about the two radioactive substances mesothorium and thorium X. Their qualities, the cheapness, and last, but not least, the good results which have been obtained by their use, seem to destine them to supplant that much more expensive metal, bromid of radium.

Mesothorium, which was discovered by Otto Hahn, has chemical qualities similar to those of radium. However, mesothorium and radium rays differ from one another in the fact that the permeability of mesothorium β-rays is somewhat less than that of radium β-rays. Besides the rapid β-rays, mesothorium possesses a group of easily absorbable β-rays, which radium lacks. This large number of soft β-rays in mesothorium explains the biologic and therapeutic differences which distinguish the two metals.

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* New York Medical Record, 1907 and 1910; Arch. of the Roentgen Ray, 1910.

Broadly speaking, however, from a therapeutic point of view, given external local application, mesothorium rays are similar to those of radium and its salts. Mesothorium, which is derived from the Brazilian monazitsand, almost always contains a certain percentage of radium according as this metal contains more or less uranium. Ordinarily, the proportion of mesothorium to radium is as 3 is to 1.

Mesothorium, which is, as a rule, used as a local external application, is applied by means of plates of varnish, capsules, glass or metal tubes, which contain mesothorium not in pure condition, but in combination with a salt, bromid of mesothorium. The modern method of application is based on a measurement of rays combined with the proceeding of graduated filtration, by means of which it is possible to choose from the total radiation of mesothorium those rays which one wishes to employ. The envelopment of the capsules, tubes, etc., retains a part of the x- and β-rays and thus acts as a filter. According as we wish the effect to take place in the superficial or in the profound strata, we can, by means of absorbing filters, separate those rays which have great power of penetration from those which have less.

The diminution of rays which is obtained by the employment of filters can be compensated for by extension of the time of irradiation, by employment of strong radio-active substances, by radiation from several points of vantage (cross-fire method). The filters most in use are plates of india-rubber of ½ mm. thickness, plates of aluminum $\frac{1}{100}$ to $\frac{1}{10}$ mm. thick, plates of lead of $\frac{1}{10}$, $\frac{2}{10}$, $\frac{5}{10}$, 1, and 2 mm. thick wadding and black paper. India-rubber is useful from an antiseptic point of view. It is permeable for hard β- and x-rays, and part of the x- and soft β-rays. Plates of aluminum have the disadvantage of forming a number of secondary rays, which may cause pigmentation of the skin. To absorb them it is expedient to place 20 pages of paper or 1 cm. of wadding between the skin and the lower part of the filter, a proceeding which, at the same time, eliminates the x-rays. Plates of lead permit the employment of the isolated ultra-penetrating rays (hard β- and x-rays), as they absorb the x- and soft β-rays.

According to the sort of filters employed, we distinguish weak, moderate, and strong filtration. The greater the distance from the skin, the stronger must be the filtration; the stronger the filtration, the longer the time of application. In the case of superficial epitheliomata, which have a thickness of less than 1 cm., mesothorium radiation without a filter, or with a weak filter, applied for several hours, will have a strong destructive effect, while for the irradiation of tumors of greater depth only hard β- and x-rays should be employed, which are not so numerous as the x-rays, and may be applied for as long a time as two hundred hours. Weichmann's instrumentarium, containing a round large and oval small capsule and several sorts of filters, seems to me to suffice for local application.

Sometimes it is of advantage to employ instruments which render it possible to irradiate organs which would otherwise be inaccessible. Thus Czerny and I have constructed an instrument* which is serviceable for the treatment of cancer of the esophagus. It consists of a graduated stomach sound through which a graduated mandrin, 2 mm. in diameter, can be drawn. At the point of the mandrin, which is double the length of the hollow sound, there is a removable celluloid capsule, 4 cm. long and 6 to 9 mm. wide, tapering off at the bottom. It contains bromid of mesothorium.

Sounding is performed as follows: The sound is introduced armed with mandrin and celluloid tube. After the obstacle has been reached, by dint of palpating and rotating manœuvers, the tube is carefully introduced as far as possible into the stenosed passage. The hollow sound is withdrawn over the mandrin, which serves to fix and later to withdraw the mesothorium apparatus. This instrument can remain in place for as long as two hours without causing appreciable phenomena of irritation. It is introduced at intervals of from three to four days, in order to prevent mediastinitis.

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*This instrument is manufactured by Fredrich Dröll, in Heidelberg.

As to the histologic changes caused by mesothorium irradiation: As a rule, in six or eight days fibroid tissue is formed; at the same time no appreciable change has taken place in the tumor cells (in the case of cancer or sarcoma). The formation of fibroid tissue increased day by day; it is the chief phenomenon caused by the irradiation. In this fibroid tissue we find numerous newly formed capillary vessels. The changes in the cancer cells—the so-called formation of vacuoles—appear later, about ten or twenty days after irradiation. It seems as if the fibrous tissue, growing rapidly, divided the carcinoma nodules into numerous small groups of cells, which again are divided into smaller groups and at last disappear altogether. In some cases I noticed necrosis where irradiation had taken place; in others, a remarkable infiltration of leukocytes, primary to the formation of fibrous tissue. In treating tumors with mesothorium we must be especially careful to avoid the least irritation.

Failures in this treatment are very often the result of insufficient irradiation. The Samariterhaus possessed about 250 mg. of bromid of mesothorium, which the bounty of the Kaiser Wilhelm Akademie der Wissenschaften, die Deutsche Gasglühlicht-Actien-Gesellschaft (Auergesellschaft), and the firm of Knöfler & Co., Plötrensee bei Berlin, had placed at our disposal.

During the past year, while we have been making use of these preparations, I have had occasion to observe a number of cases in which mesothorium influenced the growth of malignant tumors in the most favorable way. However, the time of observation is too short to enable me to speak of lasting effects. On the whole, about 250 patients suffering from malignant tumors were treated with mesothorium, and in more than 50 per cent. the effect was a good one. In 5 cases of stenosis of the esophagus caused by cancer, sounding with the mesothorium sound restored the permeability of the esophagus to such an extent that even solid food could be introduced, and gain in weight, and marked improvement in the patient's general condition were the result. In 78 cases of recurrence of cancer of the mamma not only did superficial nodules in the skin disappear, but likewise subcutaneous nodules of the size of a walnut. In 4 cases of recurrence of cancer of the mamma operated on we placed bromid of mesothorium in the wound after operation for prophylactic reasons. These were cases which had had several recurrences, and in which it was dubious whether radical extirpation of the tumor was possible. The wound was sutured in such a way that the tube could easily be removed after twelve hours (in one case). Generally, increased secretion took place in the wound, so that the process of healing was considerably prolonged. Recurrence has hitherto (after six to eight months) not been noted.

Thirty cancers of the face—12 epitheliomata of the lips—were subjected to our treatment. They were almost all favorably influenced by mesothorium; the carcinomatous ulcerations necrotized in a short time (in these cases bromid of mesothorium would be applied without a filter), and healthy granulations were formed.

We had 12 patients suffering from carcinoma of the tongue; 3 of them were dismissed in a considerably improved state of health. In 2 cases, I am sorry to say, it was not possible to confirm the clinical diagnosis by histologic examination. Seven inoperable tumors of the pharynx, which are so difficult of access, sometimes were markedly improved by this method of treatment, while tumors of the mucosa of the cheek and malignant tumors of the mucosa of the mouth were, as a rule, refractory, sometimes even showing a tendency to become more malignant than they had been hitherto.

I cannot here give the very interesting details of casuistry; a short time ago some of them appeared in a paper written by Czerny and myself for the Münchener medicinische Wochenschrift; a second more detailed paper is to follow.

Thorium X, which was kindly placed at our disposal by the Deutsche Gasglühlicht Gesellschaft (Auergesellschaft), is a product of transformation of mesothorium, and is used dissolved in normal salt solution. According to the Auergesellschaft, 1 c.c.

of this solution contains about $\frac{1}{1000000}$ mg. of the element thorium X, and possesses an activity of about 1,000,000 Mache units (1 Mache unit is equal to about 80 volts per hour in the Fontaktoscope of Engler-Sieveking, giving a capacity of 12.4 cm.). The activity of thorium increases by about 10 to 20 per cent. during the first day after its production by reason of the formation of thorium A, B, C, D. Then its activity decreases daily by about 17.5 per cent., so that in three or four days half of it is gone. Irradiation consists of x- and slow β-rays only thorium D has x-rays. The strong effect of this preparation is evidently caused by the formation of thorium emanation, which somewhat deviates from the place of deposit, and even in falling to pieces emits powerful rays and forms products of the same quality.

The application is either intratumoral, intravenous, or both. I do not know whether tumors have ever before been treated with intravenous injections of radio-active substances, but I do know that, on the whole, the intravenous injections of thorium did not inconvenience the patients, and that in a number of cases, especially when the tumors would otherwise have been inaccessible, they had an unmistakably favorable effect.

The technic of the intratumoral injections of thorium is simple: It is necessary to introduce the solution undiluted and into several points of the tumor, at intervals of six to eight days, because of the reaction, consisting of pain, reddening, and swelling, which generally sets in.

In these intravenous injections it is necessary to introduce thorium in diluted solutions of 1 part of solution of thorium (activity, as a rule, equal to 1,000,000 Mache units) diluted with 10 parts of normal salt solution.

On the day on which the injection is made the patient must be kept as quiet as possible, and on the following days bowel movements must be insured by the use of laxatives or enemas, as thorium given intravenously or intratumorally (the latter more than the former) irritates the mucosa of the intestines, especially of the great intestine. It is necessary to purge continually, in order to prevent the thorium X contained in the feces from staying in one place for any length of time, and there causing irritation.

The second intravenous injection should never be given earlier than a week after the first. Then we may assume that the patient has recovered from the effects of the injection, which often causes nausea, loss of appetite, and faintness during the first few days.

In cancer of the esophagus and of the intestinal tract generally one can give thorium X solution absorbed by pulverized silicic acid and mixed with sugar as pap, or in the form of pills, as described by Werner and Hessel. Pastes and plugs for caverns in tumors, caused by necrosis, can be made of the same mixture minus the sugar.

The histologic changes produced by injections of thorium X do not differ much from those produced by mesothorium. Here, too, irradiation does damage to the specific parenchyma, causing necrosis, maceration, cytolysis. The damaging effect of thorium X on capillary vessels sometimes leads to hemorrhage. Later on reaction of the stroma sets in—more in carcinoma than in sarcoma. First, we see infiltration with lymphocytes, then proliferation of fibrous cells, and then transformation of this newly formed fibrous tissue, with its great number of cells and its infiltration, as in chronic irritation, into adult fibrillary fibrous tissue. This last stage of complete disappearance of the parenchyma and formation of fibrous tissue is not always reached; the regressive changes in the parenchyma and the vicarious proliferation of fibrous tissue can come to an untimely end; sometimes the changes are even limited to an unimportant lymphoid reaction in the stroma.

In the Samariterhaus we have treated 206 tumor patients with thorium X; 58 were relapses of carcinoma of the mamma, 9 carcinoma of the esophagus, 14 relapses of carcinoma of the rectum, 10 malignant lymphomata, 6 branchiogenous carcinoma, etc. In 40 per cent. we could trace a favorable influence of thorium on the tumor; true, sometimes not very great, but always noticeable; in 20 per cent. we had results very much better than those usually produced by radium. We must always bear in mind that these were all advanced cases of malignant disease, in which we could not look for more than a slight improvement.

I will not here give details of casuistry, but I will mention a few specially prominent cases.

A woman of fifty, whose right mamma had been amputated on December 10, 1907, and who had had an operation for a local recurrence in 1909, came to us in January, 1912, with three subcutaneous nodules of the size of a pigeon's egg; one was situated in the upper, the second in the lower, fossa clavicularis, the third in the axilla. We gave her intratumoral and intravenous injection, of thorium X (all in all, 8,000,000 Mache units). At the same time she was treated with Röntgen rays. The nodules have completely disappeared, so have the pains from which the patient suffered at the time of her admission to the hospital.

In another case of recidivation of carcinoma of the mamma consisting of nodules in the scar and carcinomatous exudation of the pleura, retrogression of the nodules and the exudation was noticeable after irradiation with mesothorium and intravenous injections of thorium X (4,000,000 Mache units in all). The patient, who also complained of great pain and loss of breath, now feels quite well. Exudation of the pleura, usually a crux in the treatment of recidivations of cancer of the mamma, could in several cases be brought to a standstill, or even disappear altogether through the agency of intravenous or intrapleural injections of thorium X.

A case of inoperable sarcoma of the tonsils with metastatic swelling of the glands at the point of divergence of the carotis interna and externa showed marked improvement after a course of irradiation with mesothorium and intravenous injections of thorium X (1,000,000 Mache units in all). The tumor has greatly diminished, and the swelling of the gland has disappeared entirely.

Malignant lymphomata were also benefited by our mode of treatment. Further details will be published in our article in the Münchener medicinische Wochenschrift.

In mentioning the good effects from treatment with mesothorium and thorium X, I have intentionally avoided the word "cure." The perusal of the literature on radium, as employed in medicine, shows that most of the tumor patients had recidivations or died not long after treatment. The time which has elapsed since our patients were dismissed from the hospital is too short to speak of anything but transitory improvement; only those patients can be said to be cured who have had no recidivations during a period of five years or more. But transitory improvement is something worth while in the treatment of malignant tumors.

We must not forget that in the greater number of the cases named combined treatment was employed (irradiation by means of mesothorium and Röntgen rays, injections of cholin, salvarsan, etc.). However, my experience with Röntgen rays, injections of cholin, salvarsan, etc., leads me to assign the chief rôle in the production of the effects named to mesothorium, and especially to thorium X. Of course, in operable tumors treatment with mesothorium or thorium X is not to supersede or displace the surgeon's knife; it can only be a supplementary method of radical operation; but for inoperable tumors it may form an independent mode of treatment.

Improvements in the technic and of the instruments employed will probably do much to further this new method of treatment. In any case the results we have obtained hitherto are an incentive to go on with it with all the energy we can muster.

Dr. Murphy: I know I express the sentiments of those present concerning the pleasure we have had in listening to this able presentation of the work that is carried out in the most celebrated cancer institute in the world. There are many cancer institutions, as you know, but most of them are carried on on the advertising and paying plan. The cancer institute at Heidelberg is in charge of Professor Czerny, and associated with him is Professor Caan, who lectured to you this morning. Professor Czerny was originally at the head of the surgical department in the University of Heidelberg. His name is a household word in the surgical world, and in every surgical amphitheater you hear of the Czerny-Lembert stitch, so that you feel you are familiar with him. He has been one of the leading surgical minds who succeeded the great Billroth. He was Billroth's student, and I think he cut as much of a figure in the surgical world as any student Billroth had.

The work that is being carried on in Heidelberg, and which has been outlined to you this morning, shows that it is yet in the experimental stage; but we cannot help but admire the candor with which Professor Caan has presented his work, claiming nothing more for it than that it produces definite changes in the relationship, first, between the connective tissue and the malignant cell structures; second, in the fragmentation of the malignant tissue and its penetration by the connective tissue; and, third, in the final disappearance in many of the localities of the malignant cells. I think that presents the very important cellular changes that occur, as described by Professor Caan in his lecture, which I had never heard before. It is, as you have seen, practically along the same lines as the results obtained by the *x*-ray and later by radium, but it appears to be more active, more penetrating, and possibly will in the future lead to other investigations in that line which may result in bringing about a cure. You notice that Professor Caan avoided the use of the word "cure," and that was a very good thing to do, because it is used so frequently and it has deceived us so often that now every man, in talking about cancer, takes the precaution to say that these are the changes observed—think for yourselves, and draw your own conclusions or deductions. We do not speak of cures. We speak of cellular changes, as the doctor did, that are demonstrable with the aid of the microscope, and of gross changes that are demonstrable in the disappearance of the nodules, at least for the time being, so that it is not to be interpreted as a cure. It is to be interpreted as a means or force that produces changes in the superficial carcinomata; that apparently in the least superficial cancers active changes are produced by these agents to which our attention has been called, and in the deeper glands they produce less active changes, and what Dr. Caan has said concerning these agents appears to be very important. We know that in the use of a drug so well known as tuberculin it is the least understood drug of all in the whole range of the pharmacopeia.

It is the one in which there is probably greater divergence of opinion than any other agent in use at the present time. Why? Because the dosage of it is not understood and is not appreciated. That is the ray of hope I see in the doctor's discourse this morning, that we may learn to dose it so that it will have more effect in retarding the advancement of the growth, if it does not cure it, and when we speak of the cure of cancer, I think retarding the fatal termination is the term that ought to be used in speaking of all the methods, whether it is the knife or whether it is some other means, because there are so few really cured cancers. If we will advance along these lines, we may hope some day to arrive at something that will be considered a cure.

I wish to thank Professor Caan personally for his kindness in coming here this morning and speaking to us. [Applause.]

SALPINGITIS—PELVIC INFECTIONS

HISTORY

Patient, a female, married three years, came to Mercy Hospital because of severe pains in hypogastrium and hemorrhages from uterus.

Present trouble dates back to seven months after her marriage, when for the first time she began to have menstrual trouble. Previously menstrual periods were regular, every four weeks, lasting seven days, three or four napkins soiled daily. Since trouble began she has intermenstrual hemorrhages continuing from end of one period to beginning of the other. Quantity of flow is variable—sometimes soils only one napkin and sometimes several. For this trouble she was treated by having her uterus packed with gauze, but failed to get relief. August 6th she had a severe intermenstrual flooding, and she had to remain in bed for a few days, she felt so weak.

August 9th she went to a doctor, who examined her and told her she had a displacement of her uterus. He packed her vagina tightly with gauze at 11 A. M. About 2.30 or 3 o'clock that same afternoon she complained of severe pains in hypogastric region. About 7 P. M. she had a slight chill. The pains became so severe that she removed the packing herself at 11 P. M. Had emesis three times during the night. Pains have continued ever since—sharp, shooting pains, occurring over whole of lower abdomen and up left side to her lower ribs. Has had drenching sweats every night, and a temperature of 100° to 101° F. Has kept her bowels open with medicine, but each bowel movement causes her great pain. No bladder symptoms.

Seven years ago she had three attacks of appendicitis in three weeks. Had appendix removed after the third attack. Wound healed by primary union. Her appetite was always good until

August 9th; since then can take only weak tea; nothing else remains in her stomach. For the past two years she has had occasional fainting spells, no spasms, and regains consciousness in about two minutes. Urinalysis negative. Heart and lungs normal.

COMMENTS AND OPERATION

Dr. Murphy: You say the uterus was packed with gauze without effect. For what?

The Intern: To straighten the uterus.

Dr. Murphy: As I understand it, she had a hemorrhage and her vagina was packed. Where is the history of the hemorrhage?

The Intern: The doctor packed the uterus to stop the hemorrhage.

Dr. Murphy: What was the cause of the hemorrhage?

The Intern: She says the uterus was displaced.

Dr. Murphy: What was the condition of the appendix when it was removed?

The Intern: She does not know.

Dr. Murphy: How long ago did she have the inflammatory condition which she connected with the hemorrhage?

The Intern: Seven months after marriage.

Dr. Murphy: How long is it since she was married?

The Intern: Three years ago, and seven months after her marriage she began to have a continuous hemorrhage, slight in amount, but continuing from one period to the other.

Dr. Murphy: What else?

The Intern: Slight pains.

Dr. Murphy (to Dr. Golden): What is your recollection of that part of the history, Dr. Golden?

Dr. Golden: She was married three years ago. This hemorrhage was intermenstrual, and not continuous at first, but more recently it has been more continuous. Then she was packed to stop the hemorrhage. Following this packing she had a chill with high fever.

Dr. Murphy: How long after the packing was it before she had a chill and fever?

Dr. Golden: The next night, at 11 o'clock. As I recall it, she was packed in the morning, and that night she had the chill and fever.

Dr. Murphy: How many hours intervened?

Dr. Golden: About thirteen, I should say.

Dr. Murphy: What is the matter with her?

The Intern: She has an infection in her pelvis.

Dr. Murphy: What disease would you think it was, if I used the expression, "a man had an infection in his lower extremity"?

The Intern: Probably a cellulitis.

Dr. Murphy: No. Answer my question. What would you think was the matter with the patient if I said "a man had an infection of the lower extremity"? Would you think he had a paronychia of the great toe? a tuberculosis in his ankle-joint? a Neisserian infection in his knee? an infection of the bursa of his patella? an osteomyelitis of the end of the femur? or a metastatic arthritis in his hip? Which would you think he had?

The Intern: He might have any of them.

Dr. Murphy: When "pelvic infection" is referred to in that broad way, it does not mean anything definite. There is no occasion for using such a loose expression. Infections in the pelvis are distinct, clean-cut, and easily identified, and they are as distinct in their treatment, depending on the different locations, as would be the various lesions I have mentioned in connection with the lower extremity. That is the point I want to bring out. What is the matter with her?

The Intern: She has a collection of pus in her pelvic cellular tissue.

Dr. Murphy: Where?

The Intern: Extending from the tubes. It may come from salpingitis.

Dr. Murphy: Where did the infection originate? What has happened to this woman? What was originally the matter with her?

The Intern: She comes in with a history of Neisserian infection.

Dr. Murphy: As I understand it, there is no history of Neisserian infection. That I did not go into myself, and I have no positive information about it. She has been married over three years, and seven months after marriage she began to have a flow which was more or less continuous. It was an intermenstrual flow.

THE INTERN: A continuous discharge.

DR. MURPHY: Then she is flowing all the time? No, she flows a little at a time. She flows for several days—and it is continuous, according to the statement of the intern. What happened when the doctor treated her? He packed the uterus, and what happened?

THE INTERN: There was a decomposition of the discharge.

DR. MURPHY: I would not be quite as severe as that on the doctor. There was probably infection present in her vagina and in the uterus at the time, and he packed it with the blood and infective material retained in there, and that did what to the infective products?

THE INTERN: Put them under pressure.

DR. MURPHY: A matter we have always been accentuating, and whenever infective products are held under pressure, what happens? Absorption into the lymphatics, constitutional manifestations. In order that we may get a complete and clear concept of infections of the pelvis I have prepared an outline this morning here on the board of the various types of pelvic infections, how they are developed, how they spread, how you can differentiate one from the other.

INFECTIONS OF THE PELVIC ORGANS AND TISSUES

I. ATRIA OF INVASION:
    Vagina.
    Cervix.
    Uterine surface.
    Tubes.
    Peritoneum.
II. ROUTES OF SEPTIC TRANSMISSION:
    Lymph-spaces.
    Lymph-vessels.
    Blood-vessels.
    Continuity of tissue.
  (A) In parturition.
  (B) In abortions and miscarriages.
  (C) From vaginal infections from below.

(D) From above—from abdomen or through circulation.
- (A) Infections in parturition:
  - Vaginal lacerations.
  - Vulvitis and paravulvitis.
- (B) Infections in cervical lacerations:
  - Pelvic cellulitis.
  - Pelvic lymphangitis (with or without abscess).
  - Pelvic thrombophlebitis.
  - Subperitoneal cellulitis: Acute, virulent, fatal.
  - From operations.
- (C) Infections of uterine surface:
  - Placental base.
    - (*a*) Thrombophlebitis.
    - (*b*) Cellulitis of broad ligament.
    - (*c*) Endometritis—acute, infective.
  - Mucous surface:
- (D) Secondary infections from tube:
  - (*a*) Through ostium into pelvic peritonitis.
  - (*b*) Through lymph-vessels into the broad ligament.
- (E) Infections in abortions and miscarriages:
  - Thrombophlebitis.
  - Acute septic peritonitis.
  - Subperitoneal cellulitis.
  - Intramural infections.
- (F) Vaginal infections from below
  - Neisserian.
  - Pneumococcus.
  - Pyogenic.
  - Tuberculous.
  - Colon bacillus.
- (G) From above:
  - Tuberculous.
  - Carcinoma.
  - Pneumococcic and allied pus infections.

The atria of invasion are the vagina, cervix, the uterine surface, tubes, and peritoneum, all distinct, clean-cut, separate tissues, easily separated from each other. Now, then, when these are infected, what are the routes of transmission from the primary infection? The routes of advancement from the primary focus are: (*a*) The lymph-spaces; (*b*) the lymph-vessels; (*c*) by the veins and arteries, particularly the former by thrombophlebitis; (*d*) by continuity of tissue. When infection occurs with parturition, where does the infection take place? There are many types of infection with parturition, depending on the tissues that are involved in the infection. The most common type is the one I will mention first, namely, vaginal laceration. Laceration of the perineum and lacerations of the vagina mean what? These are surgical lesions in a zone that it is fearfully difficult to keep aseptic, and the only wonder is that we do not have more infections following these surgical lesions. The reason that we do not is not because the field is kept aseptic or sterile, but because nature prepares that field by the coffer-damming infiltration edema and swelling which precede parturition. The field is prophylactically taken care of and offers a high grade of resistance; therefore you rarely have infection of the cellular tissue around the vagina or around a perineal laceration.

What is the next field of infection? The next and most common field of infection following that is laceration of the cervix. The cervix of the uterus is more richly supplied with lymphatics than any other portion of the genital tract. Wherever you have a tissue richly supplied with lymphatics you have a tissue that admits of frequent infections and rapid transmission of that infective material. The lacerations of the cervix are the most common atria of admission of infection into the pelvic cellular tissue. Whenever infection takes place from a laceration of the cervix where does it go? Just where the cervical lymphatics go—into the cellular tissue of the broad ligament. Cellulitis occurs in the cellular tissue of the broad ligament when you have a laceration of the uterine cervix, just the same as you can have a cellulitis occurring in the forearm as the result of finger infection.

When you have a laceration of the cervix we may have a virulent type of infection, the streptococcic type, and there are only a few of us who remember when it so frequently occurred after exposure to scarlatinal and diphtheric infection. Infection with the streptococci means what? It not only passes into the lymph-spaces, but passes right along through the lymph-channels and lymph filters into the circulation, and the patients die from what? From infection in the cellular tissue of the broad ligament and from infection in the subperitoneal cellular tissue behind and in front of the peritoneum. When you open the peritoneal cavity in that class of cases you find only a little fluid—simply a few flakes of lymph. You find what the late Dr. Fenger called a dry peritonitis. Many of you have not lived through this, and you do not know what that means, but you must have an idea in order to get a proper and clear picture of the pathology in the pelvis.

Why is there not a lot of pus in the peritoneum in these cases? Because the infection is never in the peritoneum, but in the lymphatics beneath it, from which these patients die on the second, the third, or the fourth day. When you cut that peritoneum, you cut into an infiltrated, soggy tissue, and the flakes of lymph are on the surface. It is a dry peritonitis, resembling the redness and edema that occur in the unbroken skin overlying a subcutaneous phlegmon.

When the infection is not of streptococcic origin, but of staphylococcic origin, the staphylococci do not pass through the lymph-spaces rapidly; staphylococci pass through the lymph-spaces slowly; they are often arrested *in loco;* they produce circumscribed abscesses, and the patient has a circumscribed pus accumulation in the broad ligament or in the cellular tissue of the peritoneum.

Again, when the infection is carried into the broad ligaments, with its many large veins, present during gestation, a phlebitis or a paraphlebitis may occur,—a septic phlebitis, I mean,—forming a thrombus and even emboli, and these septic emboli may be carried through the patient's system. You may have the venous type. Here you have a thrombophlebitis, but you may have a lymphangitis and not a thrombophlebitis. Here we have the subperitoneal variety which I have just described. That comes from what? From a laceration of the cervix, with the admission of infection at the time of parturition or during an abortion. Lacerations of the cervix are not at all uncommon in abortions, but infections of that type are much less frequent in abortions. These are the infections that come from lacerations of the vagina and from lacerations of the cervix.

Let us come to the next type of infection that occurs on the uterine surface. The most common position in the uterine cavity for serious infections to take place is on the placental base. When it does take place on the placental base, what has been separating the blood of the mother from the placenta? That thin layer of membrane. In the case of a virulent infection the microörganisms penetrate the membrane and you have infection in the vein, followed by thrombophlebitis, septic emboli, septic infarcts, endocarditis, and death. When you have an infection that is less virulent and the microörganisms do not penetrate the membrane, they may be transmitted through the lymphatics into the broad ligament, causing edema and swelling of the broad ligament, associated with a chill, but never associated with septic emboli. This type is rare because there is such a sparse lymph-supply to the uterus above the cervicocorporeal junction.

The next variety of infection of the uterine cavity surface, without transmission beyond that, is seen in cases of diphtheria, with the formation of a diphtheric membrane, and its sloughing and necrosis. Fortunately, that type does not occur now at all. Then we have infection on the surface of the uterus passing on upward to the next surface, by continuity; an infection of the mucosa of the tubes. When the infection passes into the tube, it terminates by transmission to neighboring structures, (*a*) through the lymphatics that lead from the tube back again into the broad ligament, or (*b*) through the lymphatics leading down in the direction of the ovary, but not into the ovary, into the broad ligament. In another type of infection the infective material passes along the surface of the fimbriated end of the tubes into the pelvis, causing the only true type of pelvic peritonitis, which is associated with parturition or with abortions, except from perforations. That is a surface infection of the peritoneum, and has not anything like the virulence nor does it require the same treatment that the subperitoneal infections do. In a case of subperitoneal infection it is useless to do a laparotomy, because you cannot get into the subperitoneal cellular tissue, the locality where the pus is situated, while in pelvic peritonitis from infections from the fimbriated end of the tube drainage is of great value.

Dr. Golden will remove these tubes, making the usual laparotomy incision, going through the rectus muscle and sheath. On exposing the pelvic contents we find that the right tube is involved more than the left. It will be amputated, together with its fellow.

A few moments ago we took the intern to account for saying that this was a Neisserian infection originally, this being a late manifestation with retention of the infective material. We will now wipe out that charge against the doctor, as we find that this is a typical Neisserian tube, with a cellulitis on the right side. With a pelvic peritonitis resulting from the escape of material from the fimbriated end of the tube the infection may extend into the free peritoneal cavity. That gives you the range of infections.

Each of these infections has a definite clinical picture, which can be separated and outlined in its own particular place as a clinical entity. There are other sources of infection, namely, from operations on the cervix, which produce a condition similar to that of a laceration, and you get again, what? The same type of infection of the cellular tissue of the broad ligament through the lymphatics. You have occurring in abortions usually a sepsis on the placental base. That is produced by the man who tears off the placenta; he is usually filthy, surgically as well as morally, and sepsis occurs, extends into the uterus, and thrombophlebitis occurs frequently, or an acute septic peritonitis through the tubes rather than a subperitoneal or intramural cellulitis.

Then there are other infections that occur independently of parturition. There is the Neisserian infection, luetic infection, pyogenic infection, tuberculous infection, and we should add to these pneumococcus infections, because the latter infections from below are quite common, and particularly so in children. Then we should add a sixth, namely, colon bacillus infections, that occur in children and are very annoying to the doctor. For instance, a child two and a half years of age gets a vaginal colon bacillus infection which goes up through the uterus, and continues for a period of fifteen or twenty years, discharging pus all the time.

Let us go back and ask ourselves, What relation do these infections bear to certain clinical manifestations? A woman has been confined, and the next day or second day she has a chill, with a temperature of 104° to 105° F., complaining of severe pain in the pelvis not associated with vomiting, as a rule; if the chill recurs the next day, and the next day, and the pain increases in severity, what has happened? There is infection of the cellular tissue of the broad ligament, and these chills will continue for days and weeks, and occasionally for months. Every day or every second or third day you may examine the vagina, and you find on one or both sides a solid mass; the uterus is fixed and immovable. The Germans have a beautiful expression for that condition. They say it is "*eingemauert*," meaning, "set in masonry," so immovable is it.

What is the matter? That patient has a cellulitis in her pelvis, involving the broad ligament, from an infection extending from the cervix. These patients can have chills and fever, with the formation of a fluctuating abscess, and if you operate on that abscess, what will you do? How will the management of that differ from operating on a tubal abscess? Very greatly. One is an abscess in the cellular tissue, without a capsule, while the other is inclosed in the tube; it is practically encapsulated. In the former you have no more of a sac than in the circumscribed abscess in the thigh. If you endeavor to enucleate a sac in that type of suppuration, you will have your hands full. Why? Because it is a cellular abscess. It has no lining membrane.

If it is a tube that is infected, it has a mucous membrane for a capsule: it is an infection of the mucous membrane. There is a closure of the uterine end of the tube; there are valves in there that hold the gonococci, and a stricture is developed there with the same regularity as it is in the male urethra. In a pus-tube you have a primary infection of the mucous membrane with—(*a*) a stricture at the uterine end by perisalpingitis, occurring usually ⅝ inch from the cornu, and (*b*) a sealing of the fimbriated end by its agglutination to whatever surface it can contact itself with during the primary infection to prevent leakage into the pelvic peritoneum. If you open the tube and drain it without taking out the sac, what must occur? A refilling of the tube, because both ends are closed with a chronically infected mucous membrane between them. You see, therefore, why you enucleate the sac or tube in one case and why you merely drain the cellular abscess through the vagina in the other case. The pathology is not only different, but the tissue is anatomically different.

Let us take another picture now of infection of the veins on the placental base—infection in the uterus. The patient has been delivered; the next day there is no temperature; the following day there is probably a temperature of 99.5° F., but everything goes along nicely. The patient is practically well up to the fifth, sixth, and even as late as the twelfth or fifteenth day. Suddenly she wakes up in the day-time or at night with a fluttering heart, and she is fearful she is going to die. She has a sense of impending death. She sends for the doctor, the doctor arrives, listens to her heart action, and finds nothing except that the heart is on a rampage.

But she says she has pain in her right or left side, or over the liver or loin, so when she passes her urine there is some blood in it, meaning that an embolus has passed from the infected vein, or a thrombus in the uterus has escaped into the venous circulation and has gone up into the right heart and set up this tumult, has passed through, has been arrested, and has produced an infarct in the lung. It is this that gives pain on one or the other side. That patient never has primary pain in the pelvis. Why? Because she has only a uterine phlebitis which does not cause pain. You can make an examination of the uterus, and you will find it perfectly movable. Its mobility is not circumscribed, because the cellular tissue has not been involved at all; the pain has simply been reflected. It is one of the awful calamities that come on when you believe that the patient has practically recovered.

I remember my first case of that variety. It was a patient of my associate, the late Dr. Lee, who left the city for Europe on the day it occurred. The patient was a young woman, seen eleven days after her confinement. It was her first parturition. In the night she had a severe palpitation; she feared she was going to die. They called in a neighboring physician, because they knew I did not go out at night. The physician examined her, and fortunately he was an able man—professor of obstetrics and gynecology in the College of Physicians and Surgeons at that time. I go into these details to accentuate what they mean and how you must interpret them. In the morning I met the doctor in consultation. The patient told the story. I had been over the same kind of story with the late Dr. Fenger as an intern, and I knew then and there that that patient was doomed. At the close of the consultation I said to the doctor that I felt the woman was going to die. The doctor, who was a large and good-hearted fellow, had not been through that awful experience with sepsis that I had had at the County Hospital, and consequently he did not believe that she was going to die. I said, "Very well, doctor, you are the older man, but I feel it is my duty to tell the people what I think about the case, and it is your duty to take the patient and try and save her, as you believe you can." I told him I would come in any day and see her, but that he must take charge of the case. We told the husband, and he was very much alarmed at such a frightfully unfavorable prognosis when she appeared to be fully out of the dangers of the confinement.

Some two or three days after she had another attack, and finally she had a left-sided paralysis, an endocarditis with pain in the loin, blood in her kidney, an infarct in her spleen, embolism of both the right and left femoral arteries, and she died. That primary clot had passed through her veins from a phlebitis. It was septic. It would probably recur, because she had an infection of the valves of the heart, and she kept throwing emboli off until it led to a fatal termination. She never had any pain in the pelvis at all. That is the type of infection where the vein is involved.

Infections of the cervix, with involvement of the uterus following parturition, where the patients many times have chills and fever, occasionally are due to a retained portion of placenta, which requires removal. But if there is no retained portion of placenta and you do a curetment, you practically sign the patient's death warrant because you open up avenues for new absorption. I saw so many of these patients cureted in the early days when sepsis was present, and always with the same fatal result. If you had that type of infection in the thigh, for instance, you would not think of taking a spoon and scraping it out. You would want nature to put in all the granulation tissue and coffer-damming that it could to guard against absorption of the infective material, and thus prevent a fatal termination of the case from the sepsis. These cases are not cureted; they are kept perfectly quiet, immobilized, kept absolutely at rest, the head of the bed elevated, the vagina kept clear, and nothing done at all to the uterus. If you curet, a fatal termination is sure to ensue.

If there is a retained placenta and the infection is at the base of the placenta, not of the uterus, it is an entirely different proposition. Then you take it out by gentle, preferably finger, manipulation, and the patient is at once freed from danger.

Before I leave that part of the subject let me call your attention to the management of abortions, which are the most common causes of that type of infection. I had some good lessons from the best teacher, except one, I ever listened to on any subject, and that was Professor Schröder, in the Frauen Klinik in Berlin in 1883. He realized that these infections practically all came from without. If an abortion came on without being induced, or without having infective material carried into the uterus to induce it, his plan was to treat the vagina as you would an abdominal wound—keep your hands out, never make an examination, never use a douche, protect the vulva with an antiseptic dressing, and let the patient alone. If it were an induced abortion, which is an entirely different thing, the septic material has been carried in by trying to induce the abortion. Then, the moment you are called, you put the patient on the table, dilate the cervix slowly, and without laceration clean out the uterus of every portion of that fetal remnant. To use his own words, "You should never cross the threshold of the vagina in an automatic abortion until you are ready to go to the dome of the uterus." If the remnants are there and not infected from without, they can stay there two weeks, five weeks, and even ten weeks, and give no sepsis until the infective material is taken in, which shows how important it is not to examine or touch the patient.

That was a hard lesson to put into practice, and I tell you I suffered doing so. One morning, on calling on a patient suffering from an abortion who had been under my charge the previous day, a big burly fellow met me at the door and said, "We got a real doctor last night." I had ordered a dressing of the vagina without making an examination or doing anything else. As her pains became very severe during the night they called in a doctor, who examined the woman without cleaning out the uterus, and in four days the patient was dead. She got an acute sepsis and died from it. I merely cite that case to show you that in doing any work of that class one must take the disappointment that goes with trying to lead in that line, but it is the right thing, and has its own reward.

Now, what is the next type of infection that comes from below? The most common type is the Neisserian. Neisserian infection first involves the vagina; second, it often involves the female urethra; third, it involves the endometrium, and fourth, it extends from the endometrium into the tube. The moment the tube is infected with any type of infection except tuberculosis it adheres to anything that it comes in contact with at its fimbriated end, and seals the end to save the peritoneum. Neisserian infection is practically always a surface infection. It practically never produces a cellulitis. Why? Because the Neisserian microörganisms do not pass through the lymphatics. Occasionally you have in the male a peri-urethral abscess, and you know what a rare thing that is if the patient has not had instrumentation. Occasionally in the female you have an abscess occurring in the broad ligament as a result of a Neisserian infection of the mucosa of the tube. Now, as I said a moment ago, with every type of infection except simple tuberculosis the tube is sealed at its fimbriated end, whether it be streptococcic, staphylococcic, pneumococcic, or gonococcic infection. When it is a gonococcus infection, little valves or folds of mucosa at the uterine end of the tube retain the pus and the retention of the gonococci in pockets or fossæ causes peritubal inflammation and subsequent stricture, just the same as in peri-urethral infiltration in the male.

With infiltration in the cellular tissue there occurs destruction of the endothelial cells lining the tube; then comes the cicatricial contraction, closure of the fimbriated end by adhesions, closure of the uterine end from stricture, and infection of the mucosa beneath.

This case is a beautiful illustration of that condition [exhibiting tube removed]—you do not see the fimbriated end at all. The fimbriated end is completely covered up, rolled onto itself; Dr. Golden has torn it free where it was adherent, but the uterine end is closed by a stricture. You see where he cut it off. The pus accumulated in there, and then the tube dilated to this size. Occasionally the pus ruptures through the wall of the tube and you have an acute attack of pelvic peritonitis, but it always remains closed at the end. Once it becomes closed with a Neisserian infection, and once a stricture forms, it always remains closed.

If you have a streptococcus or staphylococcus infection of the mucosa of that tube, what happens? Edema and swelling of the mucosa at the neck; closure of the fimbriated end with organic adhesions, but as soon as the acute swelling disappears in the mucosa of the neck there is no peritubal infiltration; the tube becomes patent again, and the pus escapes through the uterus and passes away, and the recovery and freeing of the tube is very much like a recovery and freeing of the nasal passages following a cold. The lumen of the tube is established, the fimbriated end of the tube is fixed down in the pelvis to permanently seal, the tube slowly dilates through the hydrostatic effect of the fluid in it. There is developed what is known as a hydrops of the tube. If there is complete occlusion of the neck of the tube from ulceration; if the infection has been virulent—then you can have a stricture and get a larger sacculated hydrops of the tube.

In that type of tube with pus in it, there is only one method of treatment, namely, removal of the tube. In the other type of tube, dilated hydrops, there is no occasion whatever for taking out the tube. Just free the fimbriated end, make an ectropion of the mucosa by sewing the edge back, and the tube will return to normal and the patient can become pregnant. These patients rarely ever or never become pregnant when they have a Neisserian infection; pregnancy after operation is always possible when the infection is of the staphylococcic or streptococcic type and when the condition is simply one of hydrops. Hydrops never occurs following a Neisserian infection: it is always a pus-tube.

The next lesion to be considered is pelvic peritonitis. Pelvic peritonitis occurs with infections from below, first, as the result of retention and congestion due to a subperitoneal infection, with no communication with the pus microörganisms in the subperitoneal space at all. An operation does no good in that kind of infection. It next becomes infected by the passage of the infective microörganisms from the uterus through the tube, out of the fimbriated end, and into the pelvic peritoneum. A sac of this kind cannot be enucleated, as it is only pelvic peritoneum. It needs only drainage. Again, you have it occurring when the fimbriated end remains closed, and as the result of pressure and of ulceration and necrosis the wall of the tube ruptures and the pus escapes into the pelvis. Sometimes such a rupture occurs when the patient sneezes. It always produces a pelvic peritonitis. All we need to do in that form of infection is to provide early drainage, and that can best be accomplished by opening Douglas' pouch and letting the fluid flow out through the vagina.

In cases of infection in the broad ligament not associated with infections of the tube, opening the broad ligament and putting in a drainage-tube through the vagina is one of the easiest procedures. It is an absolutely erroneous procedure if the infection is in the tube. You see the reason why. The pelvic peritoneum and broad ligament abscesses have no sacs lining them; they are like abscesses in the thigh; they merely need drainage for a permanent cure. In the case of the tube a mucous surface is infected, the tube is closed at both ends, you cannot stop the infection with any type of drainage, and the result is that the tube fills again and again and must eventually be removed.

Let us go back and apply what we have said to this case. What happened? This woman had a Neisserian infection within her uterus, which has kept up the oozing, which is not at all uncommon with a Neisserian infection. When she was bleeding with this infection her cervix was packed, her uterus was packed, and the infective material was held in the uterus under pressure. It was forced into the lymphatics, set up a cellulitis with infiltration in the right broad ligament, which caused the elevation of temperature and the pains, and the other symptoms she had preceding this operation. You can see that packing under these circumstances is not justifiable. She had to be taken to the hospital and be relieved of the condition that was producing the hemorrhage. This type of hemorrhage is rarely ever fatal—often serious, but rarely ever fatal. There is no fibroid in this uterus.

Dr. Golden has removed the tubes down to the cornu of the uterus; he has made an abraded surface of the broad ligament; he is now covering that over with peritoneum so as to avoid adhesion of the intestine to the stumps of these tubes or to the abraded surface in the peritoneum. These adhesions are serious propositions, occasionally producing intestinal obstruction and ileus immediately after operation, more rarely producing obstruction after a longer period following operation.

In handling a case of this kind you work by an analysis of the clinical history; you come to a conclusion as to what the primary lesion is, what the secondary lesion is, and why and how you should operate.

Dr. Golden will close the incision without putting in drainage. Although he ruptured one of these tubes in taking it out, the infection is an old infection. In old infections the pus has lost its virulence to infect the peritoneum. When this was brought out first by the professor of gynecology in Halle, Professor Schultze, it was found that in 80 per cent. of the cases the pus was sterile in old pus-tubes. That explains why Lawson Tait, nearly one-quarter of a century ago, was able to remove pus-tubes and allow the pus to flow over everything, without even washing his hands, it is said, and all of his patients got well. He was called in England, for making this statement, the most accomplished liar in the United Kingdom,—*when he was really telling the truth all the time*,—based on the fact that the pus he was dealing with was no more infectious than so much serum would be, as was subsequently proved by Schultze.

Schultze, of Halle, cleared up the subject and showed the reason why Tait did not have trouble, namely, because the pus was sterile; that he was working in a field that had its lymph-spaces coffer-dammed and infiltrated by a chronic inflammation and it was practically impossible to get a new infection. This was particularly true in the case of pus-tubes. We could not understand it, because if we operated early in the disease and removed the pus-tubes, many of our operations would have resulted fatally. In the early eighties they got infection. Tait, however, was taking out old pus-tubes and doing anything he pleased, and his patients were getting well. Tait's cases had the field prepared for pus; there was infiltration, so that infection could not take place. On the other hand, in acute cases there was no preparation of the peritoneum at all, and the slightest infection spread like wild-fire and the patients died.

This shows you the resistance one has, and the safety with which this class of work can be done and the fact that one does not need to drain. If you opened one of the pelvic abscesses from above and found pus,—old pus,—then all one would have to do would be to mop it out with 5 per cent. phenol solution, as Championniere did in 1887, close up the peritoneal cavity, put the patient in an elevated position, and recovery always ensues. It is an entirely different proposition where you have a pus-pocket following an acute appendicitis. There you have virulent microörganisms; there you have the wicked type of infection; there you cannot with safety get along without drainage and expect to get uniformly successful results.

[The patient made an uneventful recovery, leaving the hospital two weeks after the operation.—Ed.]

METASTATIC GONORRHEAL ARTHRITIS OF THE KNEE

HISTORY

PATIENT, a male, twenty-four years of age, came to Mercy Hospital August 29, 1912, on account of swelling and stiffness of his left knee-joint. Trouble was initiated March 29, 1912, by soreness and stiffness in the joint. In the course of the next two days the joint became stiffer, and on the third day he had to use a cane to get about. Then the joint became swollen. The next day he consulted a doctor. He was given a liniment to rub on his knee, but after a week it was so much worse that he went to a hospital. It was more swollen above the patella than elsewhere. He was anesthetized, the swelling was opened above the patella by two lateral incisions, a rubber drainage-tube was introduced, and a Buck's extension put on the leg. The tube was taken out after three days and the extension was removed after two weeks. He was told that pus was evacuated when the swelling was opened. The wounds healed up rapidly after the tube was removed and did not discharge pus. After leaving the hospital his knee was very stiff and still swollen. Stiffness and swelling disappeared to some extent after a few months, but he has remained in about the same condition since. He has to use crutches, and movements of the joint are restricted about 50 per cent. One week after the onset of his trouble his temperature was 103° F. He had no acute pain, no chill, sore throat, or skin trouble, and he does not remember injuring the joint in any way.

In March, 1911, he had a Neisserian infection. The discharge persisted for about five months. He had a slight discharge when in the hospital for his knee trouble, but no gonococci were found. Had treatment with antigonococcus-serum (old), four doses, before joint trouble began; the fourth injection was given one month before the trouble started. He has had twelve doses since the knee became swollen. No chancre. Had typhoid five years ago, without any complications. Otherwise patient is in perfect health. No urethral discharge at present time. Family history negative.

Examination shows left knee to be swollen; swelling most marked above patella. Circumference of joint increased about one and one-fourth inches. On the second day after his admission, 20 c.c. of a 2 per cent. formalin in glycerin solution was injected and a Buck's extension was applied.

COMMENTS AND OPERATION

DR. MURPHY: I am trying to locate the etiologic factor in connection with this arthritis. To what is the arthritis due? The intern seems to get the etiology separated from the history. What was this man doing that he had a latent infection in his urethra? What was he doing the ten days preceding the first manifestation of this joint trouble? Don't you remember that he fell into a creek and got wet?

THE INTERN: He did not tell me that.

DR. MURPHY: He remembers all he was doing. He was unloading coal in Iowa. Where was his exposure? Jumping from coal-car to coal-car and working in a strained position, in a season that is neither midwinter nor midsummer—a dangerous season for the precipitation of metastatic infections. March is one of the most treacherous months we have. What happened? He was incapacitated entirely inside of three days with a large swelling of his knee. The temperature in seven days was up to 103° F. Was that tuberculosis? Not at all. That was an acute metastatic infection in his knee, and probably from his urethra. He said he was having a fresh discharge from the old trouble when he was admitted to the hospital, which is just what does occur in these cases.

The metastatic infections in the joints that occur in association with urethritis occur under two conditions: First, they occur from the eighteenth to the twenty-second day after the primary infection; nineteen out of twenty cases occur in that time.

That is not a coincidence or an accident. To have that uniformity going on for years and years is more than a coincidence or an accident. There is a definite time when, with this type of infection, the microörganisms are admitted into the circulation and are carried to their positions of metastases. That is the third week period practically, and that differs from the metastatic streptococcus infection in that the latter is precipitated in its metastases usually within forty-eight hours, rarely ever later than seventy-two hours, after the initial infection.

Then there are the other varieties, like the staphylococcus and the grip infections, that occur about the ninth day; pneumonia, from the eleventh to the fifteenth; and typhoid metastases from the second to the fourth week; the lower types of infection run a little longer time before they are precipitated in the joint. These metastases occur with almost as exact regularity as the secondary infections or the perforations or the hemorrhages in typhoid.

Each type of infection seems to have a definite time for metastases to develop. A definite period of time elapses from the primary infection before metastasis occurs. When it is associated with trauma, it comes on a certain time after the trauma.

Then the patient consults the doctor, who makes an aspiration of the knee and finds no gonococci in the fluid. He concludes that it is not a metastasis from the Neisserian infection; only in 7 cases out of 100 did even the most careful observers find gonococci in the aspirated fluid. Other observers have found them in 72 cases in 100, depending on the period of time that they withdrew the fluid after the appearance of the joint manifestation. In our experimental metastatic infections of joints we found that the microörganisms never, except the streptococci, appear in the fluid early; that a metastatic infection is an infection of the substance of the synovial membrane and not of the surface of the synovial membrane, and the secretion is a manifestation of an intramural infection of the synovial membrane, and, therefore, there are no microörganisms in the joint when you aspirate it. This fact misled us for a great many years.

This boy has an elevation of temperature. He does not remember having a chill. The rule is that they do have a chill, and quite a severe chill, even in the Neisserian metastatic type of infection. Then comes the inflammatory process. The boy's reaction to tuberculin is absolutely negative. It is a metastatic infection of Neisserian origin in the synovial membrane with advanced infiltration and contraction of the capsule.

He will be treated with repeated injections of a 2 per cent. formalin in glycerin solution to get a strong reaction. That reaction must be strong enough to overcome this infection in the neighborhood of his joint. Formalin and glycerin injections have two effects. The first is to increase the polynuclear cells in the fluid in the joint, and the second is to increase the polymorphonuclear leukocytes in the blood and in the joint. We do know that these patients get well and get well quickly with this line of treatment, if administered early in the disease.

I wish to mention another important thing in connection with the administration of the injection. We always use a tenotome to puncture the skin. The needle of the syringe is inserted in this opening. When you give the injection and produce a chemical inflammation, do not let that patient walk about. It is so hard to get people to appreciate that, because the pain is relieved materially in forty-eight hours. The patient must be kept at rest with an extension on his leg so that the joint is completely at rest during the process of repair. This is nothing new in principle. This is doing what the old Egyptians did with granulated eyelids.

After the needle is pushed into the joint we make negative pressure first. You see the fluid in the syringe. I know I am in the joint. The fluid from the joint always rises to the top of the glycerin. That is proof positive that the needle is in the joint. Inject the solution slowly. Withdraw the needle and seal the opening with collodion. Extension will be put on that leg to relieve intra-articular pressure and tension and to keep the joint surfaces from coming together.

Going back to the Egyptians, what did the old Egyptians do? They took copper or silver nitrate and set up an acute chemical inflammation, to cure what? A chronic process. And so we are just applying an old principle to the synovial membrane of the joint. That is all. It does not need to have a specific effect. What I would like to use in there is turpentine, but I have not learned the proper dosage of turpentine, because when it gets into the joint or cellular tissue it produces, in practically 100 per cent. of the cases, polynuclear leukocytic pus. You doubtless remember that turpentine pus was the last thing the antimicrobic fellow fought for. It was the last ditch in which he died. The theory was that if pus was the product of microörganisms, you could inject turpentine into the cellular tissue and have pus as a result. That is all right, but you have not virile pus. It is lymph full of polynuclear leukocytes. That is all. Turpentine will be the ideal injection material. Our experiments are going on, and we hope some day to arrive at the point of knowing how to mix it in order to get the reaction we need. We can get a greater result from it than from the use of formalin. We have used it in a few cases, but we were uncomfortable when we used it, and we stopped using it on account of the element of fear.

We will now take up the subject of the management of these arthritides. How will we manage a case of acute arthritis? Acute arthritis, as we view it, is a metastatic manifestation of an infection of some open surface. We believe that the diet has little or nothing to do with it. We believe that acute rheumatism is an acute multiple arthritis due to infection, and that the infective material is carried to the synovial membrane.

The future is going to settle, and settle promptly, whether we are right in that. We are convinced, from our observations of the relationship of arthritis to infections in other positions in the body. The reason we have failed to connect "rheumatism" with primary infection is that so long a period of time elapses between the period of primary infection in the nose, for instance, and the secondary infection in the knee. The Neisserian infection is such a uniform condition, but we have many practitioners who are still fighting us by saying that it is a coincidence of "rheumatism." No. Nobody believes any longer that a joint inflammation is a coincidence of the "rheumatism" with a Neisserian infection. They all know it is a metastatic manifestation of the Neisserian infection.

The boy we have just injected had his knee-joint opened. He had a tube put in there for three days. There was pus in the joint. No cultures were made. Finally, at the end of three days, the tube was taken out, which was the saving feature of that case. We have a well-established law that if we expose any serous surface for any considerable time to the air there occurs a necrosis of the endothelial cells and a synechia or ankylosis of the adjoining surfaces, whether it be the pleura, the peritoneum, or the synovial membrane of a joint. We have also a surgical law that we must never open and drain a synovial sac and leave it open unless we are willing to accept ankylosis as a sequence. There are some exceptions in which joints are opened and drained, and the patients recover without ankylosis, but in the enormous percentage of cases they have an organic ankylosis.

We had a beautiful illustration of that in this clinic. A young boy had the knee-joint opened in a street-car accident, and a doctor put several drainage-tubes in in all directions. What was the result? Complete ankylosis.

Another case of metastatic infection, like this boy's, was drained for four or six weeks, a complete bony ankylosis resulting. That is the only thing that should be looked for. That should be a law. There is an exception in those cases where, through the special intervention of Divine Providence, a sufficient number of microbes do not get in to produce the irritation which destroys the endothelial cells and results in adhesions of the synovial membrane. If adhesions of the synovial membrane occur in children, the result is ankylosis and ossification.

These cases accentuate the fact that one must not open a joint and drain it. Destruction of a joint from infection occurs from two essential elements: one common to infections in all positions in the body, the other specifically associated with joints. The one common to all positions in the body is the retention of infective products under pressure. Wherever acute infective products are held under pressure there is ischemia of the surface; death of the polynuclear leukocytes; the production of trypsin, and a rapid destruction of tissue.

Why do I say this? Because we all know that in the case of a phlegmon of the thigh, all you need to do is what? Cut a hole in it. You do not need to wash it out or scrape it out; you do not need to touch it, other than to take off the tension, and it gets well. Now, the same would be true in the joint if we had to deal with a connective-tissue surface. Here, however, we have to deal with a synovial surface, an endothelial-lined surface, and not only that, but, accumulating in the joint are the products of infection under pressure. Why does that not come to the surface and act practically like phlegmonous or metastatic infections in other positions in the body? Because the joint is surrounded by an inelastic, non-vascular, white fibrous capsule. It is never the seat of a primary infection; it withstands an intra-articular pressure of from 40 to 120 pounds of hydraulic pressure. Dr. Speed tested this pressure by drilling a hole into the patella, introducing a cannula into the joint, and trying to burst the capsule. But it does not burst from that pressure.

It does not necrotize from infection like the other tissues do, and the result is that the products of infection are held in the joint under great tension for a long time. What does this infective product do? It destroys the velvet lining of that joint, the endothelial layer of the synovial membrane, and when that has happened, what is the result? Wherever there is a contacting of this denuded surface, adhesions form and there occurs fibrous, then bony, ankylosis. That is the common occurrence in the joint.

There still is a second element of pressure in joint infection, namely, muscular contraction, immobilizing the joint, forcing the inflamed surfaces against each other, and it is not at all an uncommon thing for me to find, when I cut into a joint and do an arthroplasty, an area of bony ankylosis not as large as a nickel where these bones pressed against each other. If, during the acute inflammatory process, you keep on a Buck's extension, you avoid the intra-articular pressure, and if you neutralize the infective product by injection, as we do it, then you destroy the destructive elements in the joint, and in an enormous percentage of the cases ankylosis can be avoided. It is a positive certainty that all postarthritic deformities can be avoided by simple extension. By avoiding intra-articular pressure you avoid pain and render these patients so comfortable that you do not have to give and continue the use of hypodermic injections.

That should be the treatment of these patients, whether we speak of these cases as metastatic arthritis or "rheumatism." You must treat the joint and not the name of the disease.

ANKYLOSIS OF ELBOW—ARTHROPLASTY

HISTORY

Patient, a strong, healthy man, aged forty-nine years, came to Mercy Hospital on account of ankylosis of the left elbow-joint. On February 8, 1912, he fell off a wagon, landing on his left side, the weight of his body coming on his left elbow and forearm. After the accident he immediately sent for a doctor, who gave him an anesthetic and diagnosed a fracture of the upper end of the ulna. He reduced the fracture and put the arm in a plaster-of-Paris cast, with the forearm flexed to a right angle, and midway between pronation and supination. The cast was left on for four weeks, being taken off occasionally by the doctor. At the end of four weeks the cast was removed permanently and the patient carried his arm by his side. He could flex and extend it a little at this time. The elbow soon began to get stiff, and at the end of six weeks there was no motion whatever in the elbow-joint. Patient was again anesthetized and the doctor manipulated the joint, endeavoring to break up the adhesions. After this manipulation the elbow was swollen and painful for about two weeks; then the soreness disappeared and he could move his forearm fairly well. He could touch his forehead with his fingers and could extend the arm almost fully, but he could neither pronate nor supinate the hand.

At the end of three more weeks the joint began to stiffen again. For the past two months he has had no motion in the joint. About six weeks ago his doctor had a skiagram made, but he did not think that he could do anything to remedy the trouble. The skin was not broken at the time of the accident and he has never had chills or fever.

His humerus shows no evidence of being fractured. His forearm is ankylosed in the semiprone position at an angle of about 140 degrees. The upper end of the ulna is not in the normal line. There is scarcely any motion in the joint. Cannot fully flex or extend his fingers; no loss of sensation and no swelling of forearm. Had been in good health previous to accident. No venereal history.

COMMENTS AND OPERATION

The history you have just heard is typical of a fracture in the neighborhood of a joint up to the stage of ankylosis. It is also typical of the result that is obtained by forcible manipulations under anesthesia made shortly after the fracture. When you cannot succeed in getting or increasing motion by flexing and extending a joint slowly and inducing the patient to carry out motion, it is rare, indeed, that one ever succeeds in getting increase in motion under anesthesia, and making forcible flexion and extension under anesthesia, that is, in these fractures. It is an entirely different condition from the fixation that one occasionally has after a metastatic arthritis. After a fracture near a joint, if you forcibly extend the limb and pull the fragments of the periosteum loose, or whatever else you do, you increase the amount of bone production around that joint, and consequently you increase the limitation in place of increasing the motion.

My associate in my early days, Dr. Lee, had an enormous practice in this kind of emergency surgery, and it was nothing for him to have six or eight cases of fracture of the elbow-joint coming to the office at one time. I had the opportunity to take advantage of his great experience. He was a keen observer, and he treated fractures very much better than we treat them at the present time. One of his rules about managing fractures of the elbow, after he had placed the fragments in position,—and his reductions were usually good,—was to dress them in all sorts of ways, depending on how he succeeded in getting the best reduction and the best retention. He would dress a fracture of the elbow with joint in extreme flexion, the arm acting as a splint for the forearm. In another case he would dress it with the arm in the full extended position.

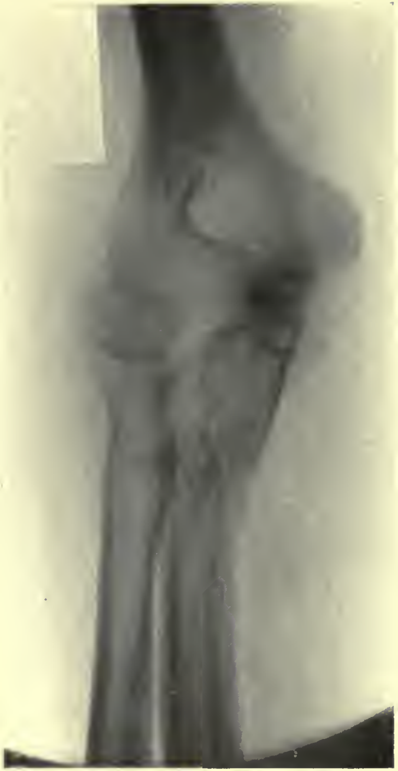

Fig. 138.—Skiagram, anteroposterior view. Shows clearly the excessive bone production on coronoid process, the fracture of the ulna, bony union between the ulna and radius, enlargement of the head of the radius,—and a fracture of the tip of the external condyle of the humerus.

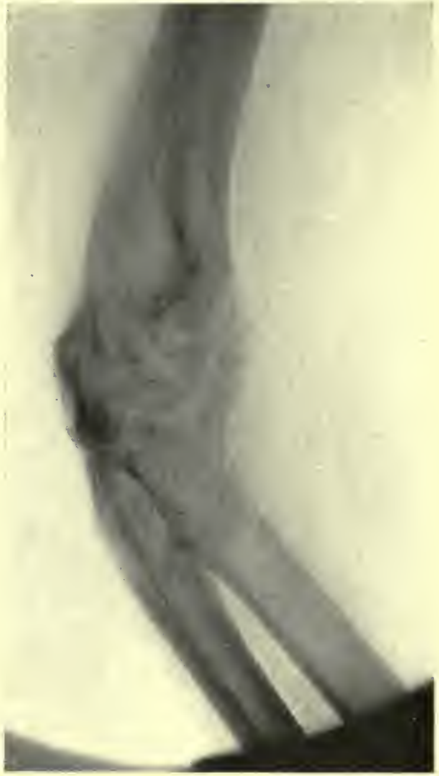

Fig. 139.—Skiagram, lateral view. The conformation of the joint is very hazy. The fracture of the upper end of the ulna and the excessive bone production on the coronoid process and tip of the olecranon are apparent.

He treated fractures, not with splints, but with the use of his cortical cells. He thought about his fractures a great deal, and he had no routine procedure in the management of them. But one of his principles, after he took off the splints, which he did on the twelfth or fifteenth day, was to begin motion that was painless, and when that progressed to the degree of producing severe pain, he said that would make the patient worse. That is exactly in consonance with our more recent observations in this type of trouble.

Let us examine the skiagram and see where this man has limitation of motion. He has a fracture of the upper end of his ulna; he also has a fracture of a portion of his external condyle. This is a class of cases that one does not desire. It is as described by the doctor, showing that the doctor diagnosed the condition correctly. What has occurred? He has an angular union here of the ulna. He has what is far worse than that—he has an excessive production of bone on the coronoid process. Furthermore, he has an excessive production of bone on the tip of the olecranon. There has occurred in this case, what occurs occasionally when there is no fracture at all, namely, ossification of the fibrous layer of the capsule. Remember, the fibrous layer of the capsule is a continuation of the osteogenetic fibrous layer of the embryonal tissue. That becomes active and makes bone in the syphilitic joint, the joint of Charcot; in the traumatisms that occur in joints, occasionally in the rheumatoid arthritis, you have ossification advancing through that capsule.

The next place where you see the same type of ossification of white fibrous connective tissue is in the muscles, namely, an ossifying myositis. These are embryologically exactly the same, and that is what has occurred in this particular case. The doctor had no control whatever over that result.

I recall one of the very great surprises of that kind that came to me in my early practice. A young girl was lying in a hammock not more than four feet from the ground. She tipped over backward and struck on her elbow. A doctor was called, examined the elbow, and stated that the child had no fracture.

This was in the beginning of the x-ray period. We examined her carefully eight weeks afterward, and while she had no fracture that we could discover, she had complete fixation of that joint. She had ossific union of the ulna and of the humerus as a result of the traumatic change in that capsular tissue, which finally led to the destruction and the ossification of the cartilage, the same as in the embryo, and to complete bony ankylosis, just as this man's case is going on to complete ankylosis.

There is another condition present here, namely, an ossification, apparently, between the radius and the ulna, just below the joint. There is not the slightest pronation or supination. The arm is perfectly fixed. In looking at the skiagram it looks as though the periosteum, that is, the fibrous layer that extends up and becomes the fibrous layer of the capsule, has also begun to ossify, and there is bony union between both bones of the forearm.

We are going to remove some of the bony deposits around these bones and interpose the fascia and fat or muscles to prevent union, and thus endeavor, through an arthroplasty, to give this man a serviceable elbow. He has ankylosis of the elbow in a position where he is unable to bring his hand to his mouth. He is unable to dress himself. He is in a lamentable condition so far as his arm is concerned. Forced flexions and extensions of the arm under anesthesia, for a time, would insure final ossification and complete bony union, so that nothing is gained by waiting longer.

The limitation of motion that occurs in the shoulder, and which produces a fixation of the scapula to the humerus, is not nearly so frequently ossific as it is in the elbow. A fibrous infiltration or a fibrous ankylosis occurs much more frequently in the shoulder than does the ossific ankylosis, but there is more pronounced interference with motion. It is not so serious in the shoulder, however. Why? Because in time the scapula gets a very great range of motion and gives one very good use of the arm, even when there is bony ankylosis between the scapula and the humerus.

This is not nearly so favorable a case for operation as a case of ankylosis that occurs from metastatic infection or from so-called "rheumatism," because the neighboring tissues are all infiltrated here, and we have a much more uncomfortable condition to deal with.

We will need to take off a large portion of the lower end of the humerus, as well as a portion of the coronoid process. In the plan of incision, therefore, which we shall adopt here we shall make two longitudinal incisions, about four inches in length, one on either side of the olecranon process. We shall take the flaps for interposition from tissue which is soft and movable, with the pedicle above, passing the inner flap under the ulnar nerve, and passing the outer flap over the end of the humerus. There is a deviation in the line of fracture. The olecranon is an inch out of plumb, but this defect we will not endeavor to correct. It is the absence of flexion and extension that concerns him. Whether that elbow is angular or not cuts no figure whatever.

I will direct my efforts first to isolating the ulnar nerve. I will free the nerve from its bed and draw it to one side, so that it will not be injured in the subsequent steps of the operation. You can expose a very long area of the ulnar nerve. All these nerves have a long central artery and some of them have a long central vein.

One of the greatest surprises of my recent European trip was the demonstration of varicose veins in the sciatic nerve. These varicose veins were as large as my index-finger, accounting for the many sciaticas which we have been treating ineffectively. I saw about twelve specimens of these varicose veins in the sciatic nerve, which were shown to us by the pathologist at Frankfort.

The next step is to expose the lower end of the humerus, the internal condyle, and the surface of the ulna by detaching the muscles on the inner side. I am doing down to and elevating all the tissues anterior to the periosteum, but not the periosteum itself. It is not a bony ankylosis. The bone has merely a projection. Now I am down on the surface of the ulna and I am going up along the exostosis that is producing the ankylosis. I am going to take off the coronoid process where it is impinging on the humerus in front, preventing flexion. I will also remove the tip of the olecranon, because it is so large that it prevents extension.

There is no bony ankylosis here anywhere between the humerus and the bones of the forearm, but there is bony union between the radius and the ulna, about two inches below the head of the radius, which is the cause of this man's inability to pronate and supinate his forearm. We will chisel that free and interpose a flap of deep fascia or intermuscular fascia from the front of the arm, so that the bony union will not recur.

I find that we still cannot put this arm in the position of extreme flexion. The head of the radius is so large that it impinges on the external condyle of the humerus. We will, therefore, remove the head of the radius completely with bone-cutting forceps, being careful not to injure the annular ligament or the pronator muscle. The ulna is free and we cut the posterior capsule so that we can look through the joint from one side to the other.

The next step is to suture the fascial flap over the external articulating surface of the humerus. This flap is taken from the deep fascia on the external lateral aspect of the arm, base downward. It is drawn into the joint and sutured to the remnants of the joint capsule. This will prevent subsequent ankylosis.

Let the records show that there had been a fracture of the ulna close to the olecranon; that there was a fracture of the radius; that there had been a fracture of the external condyle, with the production of callus in front and luxation forward of the radial head. The head of the radius was removed; the tip of the olecranon and tip of the coronoid process we also removed. A fascial flap was implanted between the side of the upper end of the radius and the ulna; a flap was also fixed over the external portion of the articular surface of the humerus; that the ulnar nerve was exposed and freed in the beginning, and was placed in the cellular tissue external to the capsule, so that it would not come in contact with the cicatricial tissue and be strangulated subsequently; that the posterior capsule of the joint was completely freed.

We have now removed all the obstructions to joint motion, as well as forearm motion. We have interposed soft-tissue flaps and we will now close the wound, using chromicized catgut for the deep sutures (this material was also used to suture the flaps), plain catgut for the superficial sutures, and horsehair for the skin. The wound is then dusted with bismuth subiodid and sealed with collodion gauze. A plaster-of-Paris cast will then be applied with the arm at an acute angle with the forearm. The arm will be kept elevated from the body by a pillow.

In about ten days we will remove the stitches and institute passive motion. The cast will not be removed permanently until after four or five weeks.

FRACTURE OF THE PATELLA

HISTORY

Patient is a young man, aged twenty, very muscular, who came to Mercy Hospital on account of a fracture of his patella (right). One morning, when coming downstairs, the heel of his right boot caught in a brass plate on the stairs and he fell forward. His knee was flexed forcibly when his foot caught, and he heard his knee-cap snap. He fell down three steps to the next landing. He did not attempt to get up and walk. He was taken to a hospital at once, and his leg was put up in splints (two long lateral splints extending from above the knee to below the ankle). He came on to Chicago last night and was admitted to this hospital today.

Examination showed the knee to be swollen, somewhat inflamed, and the patella fractured transversely just below the center. The two fragments were separated about one inch. A Buck's extension was put on after 15 c.c. of a 2 per cent. solution of formalin in glycerin was injected into the knee-joint preparatory to doing a wiring operation one week later.

COMMENTS AND OPERATION

The lack of success in all this work, as brought out by Mr. Lane, is due to failure of securing a contacting of the fragments. In this case the bone is broken transversely. The capsule was divided, as we estimated it, below the line of division of the bone, and the upper margin turned down, and is covering the fractured surface of the upper fragment, which is the most common cause of the failure of union in these cases. I have here an opportunity to demonstrate that condition.

We will expose the patella by a curved incision across the joint extending from one femoral condyle to the other. The convexity of the flap is directed downward. Raising up this skin and fascia flap, the fractured patella is at once exposed. Here is the lower end of the upper fragment, and you can see how beautifully the torn capsule has capped it over. That is why we have so many failures of union; not that there is a failure of activity of the osteogenetic forces, but the capsule has fallen in between the fragments and prevents union from taking place. I demonstrated that when I first began to practise surgery in Cook County Hospital many years ago.

There is a blood-clot in there which I shall pick out. I am endeavoring not to touch the synovial membrane with sponge, instrument, or finger. I am lifting that clot up and out without touching the synovial membrane even with my instrument. Note the very large size of that clot. It is on the end of the bone and not in the joint. If I do not touch the synovial membrane, I will not have an excessive secretion after the operation. I will not have a hydrops articuli. Here is the lower button of bone, and it is small, as is shown in the skiagram. It is turned upward and out, so that the articular surface of the lower fragment comes against the aponeurosis of the patella. That prevents the possibility of union occurring. We will raise up this aponeurosis and release the lower fragment. When I throw the aponeurosis inward this button of bone comes down. Having removed the clot and assuring ourselves that the fragments can be approximated, we will fix them by wiring.

We are ready for the insertion of the twelve-strand phosphor-bronze wire which will encircle these fragments and hold them firmly in apposition. In an article I wrote a short time ago on fracture of the patella I described the method of turning down a flap from the quadriceps tendon as a substitute for the patella. I believed that that was original with me. When I was in Rome, Dr. Bastianelli had the kindness to tell me that it had been done over there by some one else a year before. So you see, we are looking back to Rome for the modern things.

First, I am going to place in my heavy wire suture, and that wire suture is going to surround the two buttons of bone. It will not be in the joint at any time. I will pass this wire through

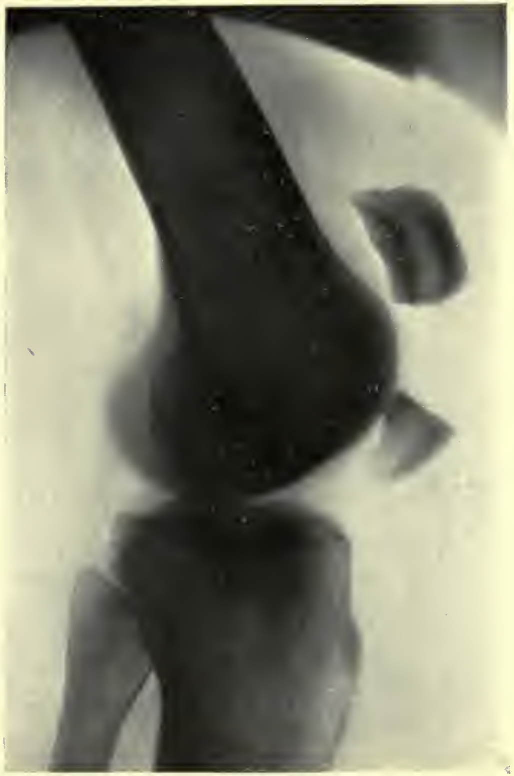

Fig. 140.—Transverse fracture of patella with wide separation of fragments. Soft tissue overhung the edges of the fragments.

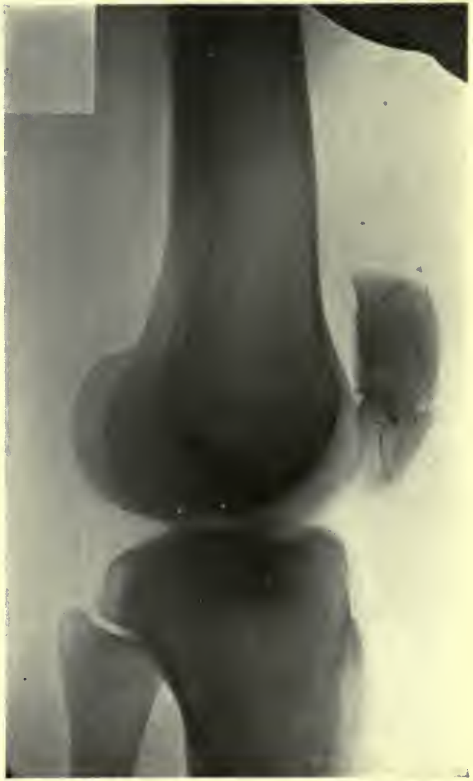

Fig. 141.—The ends of the fragments were freshened and approximated. Two encircling phosphor-bronze wire ligatures maintain approximation and fixation of fragments.

the quadriceps tendon well above the bone, going from the outer side to the inner, encircle both fragments of the patella, and emerge at the point of entrance. For security, I will insert two wires. One is inserted at a little lower level than the other, making practically two tiers of loops, so that when these wires are pulled taut the fragments will be well in apposition and held so firmly without tilting, which might occur if only one loop of wire were inserted. They are then made taut to bring the fragments into firm approximation and tied.

I mentioned Mr. Lane's name in connection with the management of these fractures. The profession has had some good results and some poor ones from Lane's plate; for the latter Mr. Lane is not to blame. You have to see Mr. Lane work to realize what real non-contacting with the tissues worked on means. These two wire loops are knotted, not twisted. This wire is almost as pliable as silk thread and will not break when knotted. I am going to sew the capsule with chromicized catgut first and turn the flaps up so that the inner surfaces of the flaps face each other. That is to prevent the capsule from going between the bone, and so as to have two smooth surfaces in contact and adhesions cannot form.

We have changed our ideas about the treatment of fractures of the patella by the open method very materially in the last few years, and we have changed them for good and sufficient reasons. Some years ago, when the open method of treatment first came into vogue, there were some painful sequences.

I believe that the capsule, as it is reconstructed, could hold these fragments in postion by itself, but I will not rely on it, as we know that catgut softens, and we know that it would take the bone a much longer time to have wire cut through than it will to have soft tissue cut through muscular contraction, and we know that in fractures of the patella there is continued traction of the quadriceps tendon or muscles from the very beginning. All that work was done, so to speak, outside of the joint. I neither touch my instrument nor the sponge to that synovial membrane. Now, then, I have two wire sutures in here, one of which may break. If it does, we have a second. That is phosphor-bronze wire. It has great tensile power, but it must be knotted and not twisted. Twisting it does not make it firm. It has one little inconvenience, and that is, that there is occasionally a little irritation when the patient strikes his knee, and it feels very much like the prick of a pin.

I started to tell you that some years ago—twenty-five or twenty-eight years ago—we began doing the open operation for fractures of the patella, sometimes with brilliant results, as was originally done by Mr. Lister, sometimes with disastrous results, as was done originally by a number of other men, and these painful results were due to the infection that followed, and these infections followed for a long time. Finally, we began to ask ourselves some questions as to why infections were more common in working on the knee than in other places, and we found the solution of that in the fact that the secretion from the joint and the irritations of the joint produced a material that had little, if any, resistance against infection; that the synovial fluid contained a lesser number of lymphocytes, the polynuclear and mononuclear lymphocytes that we find in the fluid of the peritoneum when it is irritated. In other words, that fluid has little resistance. Furthermore, we found that after the surface was handled, the irritated synovial membrane was prone to infection, and that its surface did not offer the resistance that the peritoneal surface offered. Finally, with the advance of time came a new line of treatment, and that treatment consisted also of the open method, but the late operation. In place of doing the operation in the first or second twenty-four hours the operation was postponed from six to ten days, and then, for the first time, we began to get satisfactory results from the open treatment of fractures of the patella.

We began to ask ourselves, then, why was there such a difference in the results when the operation was done on the tenth day or when it was done on the first or second day? And we believed that the reason we got better results was that the tissues immediately after the trauma were in a state of least resistance to infection; that when one waited ten or twelve days before operating, a condition had been brought about in the mean time of tissue reaction, or a granulation tissue and infiltration had formed, meaning a coffer-damming of the lymph-spaces in the tissues. That was one of the elements that contributed to the good results, and we believe that is the real explanation for the good results of the late operation, and that the absence of this tissue resistance to infection was the reason that so many cases went wrong after the early operation. If you have a synovial membrane or a joint or any tissue anywhere in the body that is injured, shortly after the injury comes the coffer-damming of the lymph-spaces, the tissue reaction, and repair. That coffer-damming puts the tissue in a condition of granulation, so that resistance is developed.

In later years we have not depended on the wound reaction to prepare the defense of the knee-joint, which is the most difficult of all the joints to operate on with safety. We have induced artificial coffer-damming of the lymph-spaces in the tissue in this case. This boy was injured on Friday or Saturday, a week ago today, and was taken to the hospital on Sunday. We immediately put him to bed. On Monday morning we injected 15 c.c. of a 2 per cent. solution of formalin and glycerin into the knee-joint to produce a chemical inflammatory reaction in the tissue and prepare the joint for the procedure which we have performed today. We also put on a Buck's extension (20-pound weight attached) to keep the joint surfaces apart and to overcome muscular contraction. We have, we believe, coffer-dammed that tissue sufficiently to care for the joint. We believe that when we look at the knee in forty-eight hours from now there will be no accumulation of synovial fluid in it: that there will be practically no irritation reaction on the part of the synovial membrane. In other words, we have coffer-dammed the lymph-spaces of the synovial membrane; we have irritated the tissues to the development of an autoimmunity or resistance against invasion by infective microörganisms.

In doing this operation, you must have noticed the care I exercised with regard to touching the tissues with anything but the instruments, and not touching the synovial membrane, and it is in following that plan in operating on ununited fractures, or in operating on fractures, that Lane has succeeded in getting such good results. I know of no one else working in this line who has been able to obtain that perfection of technic in handling fractures that Mr. Lane has attained, and that is why I think he is such an enthusiast about his method, because he has been able to get such uniformly successful results.

In this case there was not so great a separation of the fragments as we so frequently see, but there was present a condition that I think will absolutely prohibit union, namely, an eversion of the lower fragment and a tilting outward and forward, so that the articular surface of the lower fragment pressed against the patellar tendon and the fibrous capsule overlapped the edge of the upper fragment. That accentuates the significance of this as an etiologic factor in non-union in these cases more than any one other thing, I think—the interposition of the capsule. Occasionally you have not only the capsule turned in, but sometimes you have the whole upper fragment rotated, so that its fractured surface is turned upward under the quadriceps tendon. That is easily made out in the skiagram, a thing we had difficulty in making out in the pre-*x*-ray period.

My encircling wires are not in the joint or in the bone, but pass through the tendons above and below the fragments. The approximation is a bony one, with a wire strong enough to hold the fragments in apposition. We are using two wires to hold the fragments in position. I have approximated the capsule accurately all the way across. As a result of that approximation we will get bony union, or approximation of the surfaces by fibrous union, which will be as effective as the bony union. It will be just as strong and just as useful as was the result in the case in the first case I treated when I began to practise surgery. At that time I treated a patient for fracture of the patella, and that patient has been my patient for thirty odd years. He had separation of his fragments to a considerable extent. He is a fireman by occupation, and the strain on his patella is very great. To all purposes, he has had bony union. He has a good leg.

There may be an enormous separation of the fragments. In one of my cases, a patient who fractured his patella in the

Civil War, there was a separation of the patella fragments of five inches, and one would hardly know that this man ever had a fracture of the patella, so far as the usefulness of the limb is concerned. That case led me to the method of elongating the quadriceps tendon as a substitute for the patella, when I removed it for tuberculosis, and it fills a purpose just as well as the patella. If conditions were not favorable, I was prepared to make a quadrilateral flap from the quadriceps tendon, turn it down over the patella, and suture it into the tendon of the patella without opening the joint, using it as a support for his fracture.

The after-treatment of these cases consists in putting on a 20-pound Buck's extension to prevent interarticular possible compression of the bones against each other, that is, the tibia and femur, during the process of repair. This will materially aid in flexion afterward, and we will get flexion much earlier by maintaining separation during the process of repair. This is a regular treatment with us. The limb is placed in a wire trough, with 15 to 25 pounds weight attached to the leg. It relieves these patients of pain right at the start. Nothing more will be done until the end of five weeks, then passive motion will be instituted. There will be mechanical support for ten weeks. Why? Because the osteogenetic powers of a sesamoid bone are limited. That is why it takes a longer time for union to take place. The capsule at the end of that time will be firmly united, and unless the patient does something extraordinary, he will have no trouble from absence of motion in his knee-joint after he gets up. Keeping extension on during the process of repair has a striking effect. In every inflammatory condition of a joint we have infiltration of the capsule, and that is one of the great factors in inhibiting motion following inflammation. If you can keep the capsule elongated during the process of repair, you have no trouble. [The skin wound in this case was closed with horsehair, dusted with bismuth subiodid, and sealed with collodion.—Ed.]

UNUNITED FRACTURE OF FEMUR

HISTORY

PATIENT, male, aged thirty-eight years, weight 240 pounds, came to hospital August 24, 1912, on account of an ununited fracture of the shaft of his femur. Six years ago he had a Neisserian infection. No luetic history. No history of tuberculosis or malignancy in his family.

May 5, 1911, while attending to the unloading of a freight-car, the engine backed and he was caught between the platform and the freight-car, fracturing both femora. The skin was not broken, but the thighs were badly bruised. He was taken to a hospital and put to bed, his thighs were steadied with sandbags, and extension by weight and pulley applied. After a period of ten weeks no union had taken place in either leg and it was decided to operate. On July 5th a Lane plate was put on his right femur and a gauze drain inserted. The drain was removed a few days later. There was a discharge of pus which continued until November 10,1911. After the operation the limb was put in plaster-of-Paris, and the wound was dressed through a window made in the cast. Ten days after the first operation the left femur was similarly operated on, a Lane plate being inserted. The drain was removed in this case after one week and the wound healed by primary union.

Examination shows that the fracture at the lower end of the femur on the left side has united with a large amount of callus on the outer side of the bone. The right femur (fracture of upper third of shaft) has not united, but a large amount of callus has formed. The right leg is almost two inches shorter than the left, and there is a marked deformity at the seat of the fracture, with decided mobility. He cannot support his weight on this leg.

COMMENTS ON OPERATION

Dr. Murphy: This patient sustained a very severe accident, and he was in a precarious condition for many weeks, especially as to his life, having sustained a double fracture, although neither of them was primarily compound. Finally, he succeeded in getting fairly good union of one bone—in fact, very good union. There is a slight bowing of that femur, which is of no significance at all. How many days after the accident was it before the operation was done?

The Intern: Ten weeks. A Lane plate was put on with screws.

Dr. Murphy: The plate held how long?

Visiting Physician (who referred the patient): I do not know exactly when it became loose.

Dr. Murphy: Finally the femur began to bow outward and the screws became loose. There was no vitality in the tissues, but that is not the significant thing. The doctor says there was an infection. I do not think he means that infection occurred. He means that the man had a discharge of débris from the wound, and that the foreign body healed in, which is pretty good evidence that the patient did not have an infection. When you get infection with an iron plate and screws in the tissues the wound will continue to discharge as a rule until you take out the iron plate and the screws. He came out of the mild infection, if it was a mild infection, with failure of union.

The skiagram shows the Lane plate still being held by four screws, but it is loose. There is an enormous effort at the formation of callus. Nature has developed a fairly good ball-type joint. Whether I can appropriate any of that callus to advantage in repairing this fracture or not I do not know. I fear not. I fear that all that new-formed bone or callus will have to be removed when I take out the plate. Then the question is, what will I do to maintain those fragments in position? I do not know what I will do. It will depend on how I find the bone when I get in there, whether it is soft and porous, from the enormous increase in bone deposit around it, or whether it is

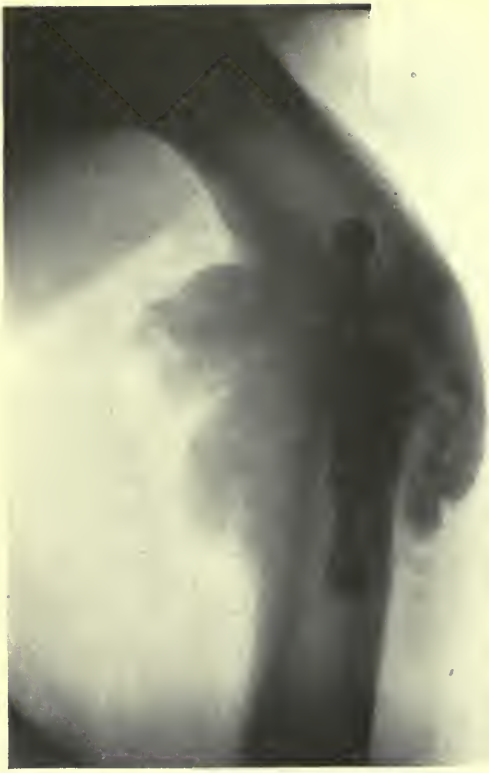

Fig. 142.—Skiagram, anteroposterior view, with four-screw Lane plate which had been inserted in the original operation performed shortly after the accident. One screw is almost out; the remaining three screws were loose, although patient's physician stated that one screw had been discharged from the wound. Therefore this screw was not searched for, and it was left in. In this skiagram it is the second screw from the top. It appears to be in the plate. When three screws had been removed, the count was apparently correct. There is excessive production of callus, with marked deformity outward. The greatest amount of callus is seen on the end of the upper fragment.

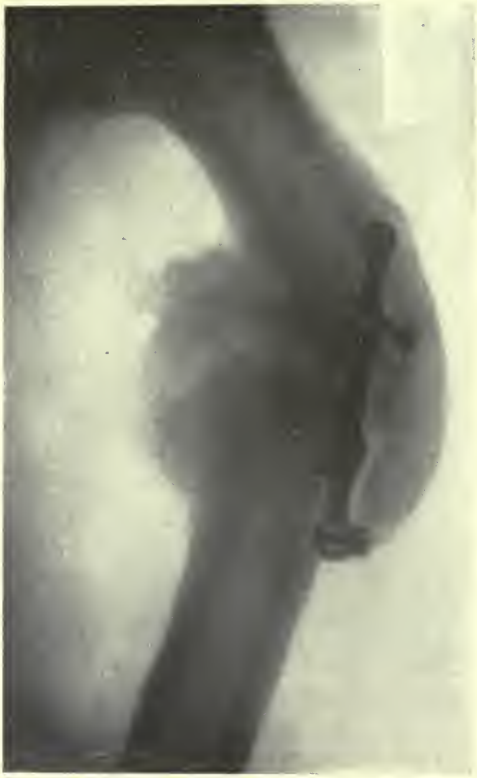

Fig. 143.—Skiagram taken more laterally, showing well how little support was given to the fragments by the Lane plate, which was too small for this case.

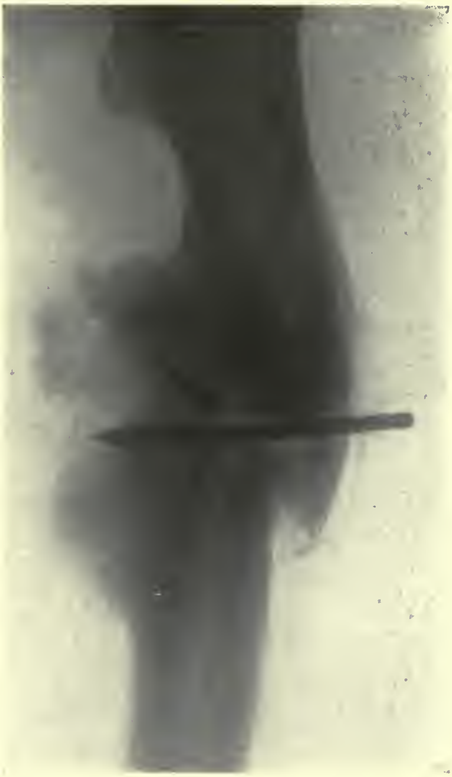

Fig. 144.—Skiagram taken immediately after the operation. The nail was driven through the bony callus on the upper fragment and through the upper end of the lower fragment. The fragments are nearly in perfect alinement, as much so as the exigencies of the case permitted. The fourth screw, which was supposed to have been extruded, is visible just above the nail.

firm and hard, so that it will retain a long bone plate. If it is soft and porous, the chances are that I will make a groove in the bone for a long distance and implant a piece taken from the tibia into this groove in the femur. That will be an interosseous splint. I want you to observe the size of this man. Look at his muscles. If I took a piece of bone—an implant—out of the tibia to put in there, I should have to remove a piece as large as my middle finger.

It is a very difficult case to handle, first, to put the fragments in proper position, because there is considerable shortening—almost two inches—and a marked bowing out of the femur. After I have put these fragments in position the question is how to hold the bone at rest until ossification takes place. If I put in an implant, it means that the leg will have to be kept at rest for three months. If I use Lane plates, it will mean fully as long a time as that. All his adductor group of muscles are contracted. The gracilis and sartorius are contracted. He has had a fifty-pound weight extension on the leg part of the time.

I will make a straight incision, about six or seven inches long, on the outer aspect of the thigh, cutting through the skin, fascia, and separating the muscles so as to expose the callus. We are down to the bone. Here is some pus between the fragments (an encapsulated bone abscess). This greatly hazards primary union following this operation—I am surprised to find it here. The bone plate is still held to the fragments by four screws. It is covered over by the callus. There are four screws in there, which we will remove in order to get out the plate. I will take out the granulation tissue between the fragments, and clean out all the débris. There is a long callus on the upper fragment as well as on the lower. The callus projects inward in the direction of the vessels. If we put in a bone transplant in the presence of pus, you will always have a failure. It will rarely heal in with infection present. In place of putting in a transplant of bone, we will get these fragments in apposition. We may put in another long bone plate and endeavor to get union without implanting a piece of bone.

Now I have the lower and upper fragments exposed and in plain view. I must get the bones into accurate approximation. I will endeavor to utilize that callus as a splint to give support. I will take off a portion of the upper fragment with the chisel, so as to get the lower fragment into position. There is no possibility of bringing that lower fragment up until I take off a piece of the upper. A V-shaped wedge will be taken out of the end of the upper fragment into which the lower fragment will be fitted and then nailed.

Let the records show that the lower fragment was displaced backward and passed by the upper fragment; that a sequestrum from the upper fragment had built a large mass of bone external to the upper end of the lower fragment; that this sequestrum was preserved; that the leg was extended until the ends of the fragments, exclusive of the sequestrum, contacted; that there was considerable difficulty in displacing the lower fragment forward, but finally it was brought forward and put into the V-shaped freshened surface of bone in the upper fragment, and then a wire nail was inserted through the callus on the end of the upper fragment into the lower fragment, to prevent backward luxation of the latter. There was great pressure on the bones, chiefly on account of the muscular contraction, which was overcome to a certain degree by traction, producing elongation and reducing the luxation. One ligature was used in the soft tissues; no drain. Skin closed with silkworm-gut and horsehair. The original bone-plate was removed, and there was pus around it. How virulent it was we do not know. Three screws were also removed. His physician states that one screw was discharged from the wound. The skiagram taken after the operation showed that the screw did not come out. This wound subsequently suppurated, as was anticipated at the time of the operation, and that was the principal reason for not inserting a bone transplant when the pus pocket was discovered in the center of the bone.—Ed.]

The limb will be placed in a Rainey splint and the leg elevated. The Rainey splint is a travois splint. It is a simple affair; it works admirably. We dress all our fractures of the neck of the femur this way, with the legs in an extreme abducted position.

FRACTURE OF THE INTERNAL SEMILUNAR CARTILAGE

HISTORY

Patient, a male, thirty-eight years of age, came to Mercy Hospital on account of trouble in his right knee-joint. Sixteen years ago he sustained a fracture of his right femur, upper third, but received no injury to knee at that time. The fracture united in good position, but there is about one and one-half inches shortening of his right leg as a sequence. During August, 1909, he struck the inner side of his knee against a chair. It felt a little sore and painful for a few days, but was not swollen or inflamed. About four weeks afterward he noticed that the knee became tired when he walked for any length of time. Although he had some pain in that knee-joint, it did not trouble him much until eighteen months ago, when, one day, he felt something in his knee slip, and he had to stop walking immediately. His knee had "locked." The knee was stiff and very painful, but he moved it gently and gradually and got it "unlocked" again. It was painful for a few days afterward, but not swollen. He had had seven or eight of these attacks, the last one ten days ago. This locking of the joint occurs if he advances his right foot quickly or when going downstairs. His knee-joint has increased in size and is becoming more painful. Since June 1, 1912, he has had to use crutches to keep his weight off the knee. For the past two months he complains of having pains in the knee at night. Has been very healthy all his life. No venereal infection. Habits good. Family history negative.

Examination.—His right knee-joint is larger than the left, and of greater circumference, about one and one-quarter inches. The enlargement appears to be caused by a thickening of the articular ends of the tibia and femur. This is undoubtedly due to a thickening of the joint-capsule. Creaking in the joint is apparent on movement. Sensitiveness over internal semilunar cartilage.

COMMENTS AND OPERATION

Is that statement about the place of greatest tenderness or sensitiveness correct? As I remember it there was practically no sensitiveness over the internal condyle, but when you get over to the inner margin of the tuberosity of the tibia, you strike the point of greatest sensitiveness. The history of this case is classic in the fact that this condition came on suddenly and followed a comparatively mild trauma; it is classic in the fact that he has had eight or ten attacks, each resembling the other; it is classic in the fact that in attempting to bring his leg forward, when he gets to a certain position and endeavors to straighten the leg, he cannot do so, and when he puts weight on the limb he has pain. Further, that after some manipulation of the joint, with the leg in a semiflexed position, he manages to get it straight and can use it again. It is classic in the fact that from these repeated recurrences of pain and locking there is great thickening of the capsule, which is one of the great dangers of this type of traumatic synovitis. Whether this synovitis is due to the loosening of a cartilage or due to infection and hyperplasia in the fatty bursa, which pushes the villous enlargements of the synovial membrane into the joint, matters not. These recurring traumas produce a thickening of the capsule—a fibrous thickening. If you wish to liken it to anything on the surface, which we desire to do, it would be like a keloid that occurs from a trauma. The capsule thickens and it becomes infiltrated. In other words, the history is typical of a luxation of the semilunar cartilage. In this case we believe that it is the internal semilunar cartilage that is causing the trouble.

Because of the construction of the knee-joint, it is strange that this lesion does not occur more frequently than it does. It is not a common disease in this country. It is a common lesion in some parts of England. It occurs frequently in men who follow athletics in this country, and it occurs with equal frequency in other countries. As an occupational disease it is quite common in the coal-mining districts of Newcastle, England. There are more cases of it there than in any other place in the world. In Newcastle the miners work in a low gallery. They work in a stooping position, so that luxation of the semilunar cartilage is almost as common in Newcastle, England, as is appendicitis. To give you an idea of its frequency, I will say that Rutherford Morrison, who has charge of the General Hospital at Newcastle, has operated in over 500 cases of luxation of the internal semilunar cartilage.

Luxation with fracture, luxation with a fringe formation in the joint, fracture with splitting and luxation and pinching, all give the same train of symptoms,—some more severe, some less severe,—but after it has once become an established lesion, there is practically nothing but surgical intervention that will effect a cure. The operation is slowly becoming rather popular, but it was a long time in doing so because we feared opening the knee.

We have two specimens removed from two patients. In one there is scarcely any change, except at the base of the cartilage, where there is fringe. The other is converted into a sponge-like mass. It does not look anything like a semilunar cartilage; it looks no more like it than a piece of cheese. It is thickened from the repeated crushings and contusions. This gives you an idea of the changes that can occur in that cartilaginous tissue as the result of repeated traumas.

The removal of the cartilage is the only means of effecting a cure. The operation, so far as the technic is concerned, is not difficult. So far as the element of danger from infection is concerned, the operation is serious. Fortunately, infection does not occur frequently, but suppuration or infection of the joint is a mighty serious matter. The joint is prepared in these cases for resistance. It is an entirely different proposition when you operate on a joint the seat of a traumatic synovitis or when you operate in a case of fracture in a joint the first day after the injury. Why? Because repeated traumas produce an infiltration of the synovial membrane and infection is much less likely to occur. In repeated traumas there is a coffer-damming of the lymph-spaces, and there is not so great a likelihood of infection occurring as there is if one has an acute infection of the knee-joint. The synovial membrane is prepared to resist infection in the first instance, and in the second its resistance is lowered.

In the removal of the cartilage we make a longitudinal incision along the inner side of the patellar tendon, and then open the joint and carefully dissect away the loosened semilunar cartilage without traumatizing the synovial membrane. Remember, in every operation in a joint keep the instruments and sponges and everything away from the synovial membrane.

The frequency of luxation of the internal and external semilunar cartilage, in comparison, is about one to fifty, according to Morrison's statistics, which are the most valuable in the world, because he had so large a number of cases. Still, that is only of relative importance to us, because Morrison's cases occur almost always from the miners working in the same position and in the same attitude, while with us they occur in athletes with the joint in all positions and in all attitudes, and we cannot estimate that which represents the average. In estimating which it is one has to analyze the individual case carefully at the time of the examination.

This patient has practically no sensitiveness over his external condyle, but he has sensitiveness over his internal condyle. This is not the most important factor. The patient says that he gets his pain in a definite spot; that when the joint locks he has pain in a definite place, namely, on the inside of the tibia, and he puts his finger on the spot. That is one of the things that leads one to make a differential diagnosis as to whether this is the internal or external cartilage that is at fault. When one of the semilunar cartilages has been enlarged and the capsule thickened, there is a wrenching of both of the semilunar cartilages, and where the capsule is much thickened, we remove both the semilunar cartilages at the operation.

We use iodin as a means of disinfecting the skin in all our work. Whether it is the best agent for that purpose we are not able to say. It is the best and the most efficient agent that we know of at the present time. Whether it is necessary to use it after one has used alcohol is a question that must be settled by future investigation and observation. However, it has conducted us safely over the road so far, and for that reason we are using it exclusively. It is giving us good results. We are not getting stitch abscesses, which is the best test of skin infection. Every one endeavors not to expose the skin during operative work, but covers it with towels and gauze. If we have to do any cleansing of the tissues immediately before the operation, we use benzin and iodin; then you can clean off the fat, and the iodin soaks in at the same time, or you can cleanse with benzin at night and use iodin in the morning. We first disinfect the skin the night before by a thorough scrubbing with soap and water and possibly with a little corrosive sublimate solution. The ordinary tincture of iodin is a bit strong. We have had two cases of dermatitis which should have been avoided. One was a severe case. The other was a mild case. But these are the only evil results we have had in about three or more years' use of it, so that we feel, so far as dermatitis is concerned, that there is very little danger from it.

I have made the incision as indicated, opened the capsule, and grasped the loose cartilage with forceps. With a slight tug it is easily removed. That joint will get along very well without that cartilage; in fact, better. The next step is to close the fibrous capsule of the joint with catgut. You can all see that white fibrous capsule. It is devoid of large vessels; it is never the seat of a primary infective lesion of any kind. It is, however, the leather bottle, as I term it, that holds the infective material in the joint when the joint becomes infected, and it withstands an enormous intra-articular pressure.

We have had some experiments made by Dr. Kellogg Speed to determine that pressure. He found that it ranged from 40 to 120 pounds before the capsule ruptured, showing the enormous pressure which it can withstand, because it is fibrous and nonvascular. This absence of vascularity prevents it from necrotizing, so that it is one of the greatest sources of danger in infections of the joint. If you have an infection in the thigh, in the cellular tissue, this tissue rapidly necrotizes, the infection travels to the surface, and the pus escapes easily through the opening made in the skin by the infection products. When you have an infection in the knee-joint, it is impossible for the infective material to escape on account of the structure and non-vascularity of the capsule.

The skin wound will be closed with horsehair. The limb will be dressed in a straight position with a heavy Buck's extension on, which is the line of treatment, practically, of all the joint infections. That relieves the intra-articular pressure during the process of repair. The limb will be kept in a straight position during the process of repair. He will not be allowed to walk on it for about three weeks and a half, and after that slowly at first. We do not apply any cast.

These cases always progress favorably. We have had about 20 of them in this clinic in the last four or five years, and all our patients have normally functionating strong knee-joints. One patient was a professional baseball player, a valuable member of a major league team. He is still playing ball, runs like a deer, and never has any discomfort in his knee.

SPLITTING FRACTURE OF THE ANTERIOR HALF OF THE LOWER END OF THE TIBIA

HISTORY

Patient, male, thirty-four years of age, came to Mercy Hospital on account of very severe injury to his left ankle-joint.

On the evening of August 24, 1912, he was standing on a ladder placed upright against a wall. He was only about six feet from the ground. The ladder slipped and he jumped off. As he struck the ground he fell backward, his right foot going in front of him. The heel of his left shoe caught in something, and the weight of his body was thrown on his left leg. Skin unbroken.

When he came to the hospital his ankle was swollen and discolored; his left heel appeared to be shortened, and a marked prominence could be seen and felt in front of the ankle-joint. His foot was not everted. Patient was put to bed with ice-bags on the ankle and foot in an elevated position. Previous history negative. The skiagram, which was made at once, showed a fracture of the lower end of the tibia (internal malleolus and part of the shaft). About three-fifths of the anterior portion of the articular surface of the tibia was broken off and slid forward and downward on the astragalus. The remainder of the tibia was luxated backward. Fibula intact. Joint ligaments torn.

COMMENTS AND OPERATION

Some days at this clinic we have extremely unpleasant cases come to us. This is one of them. It is one of those cases treated as an ordinary Pott's fracture, which, if treated as such, would cause permanent impairment of the joint. It is not a Pott's fracture at all. It looks like a Pott's fracture, but it is not.

There is inversion of the foot, which is never present in a Pott's fracture. In Pott's fracture there is always eversion of the foot. A Pott's fracture, you remember, occurs as the result of a forcible eversion of the foot, and never from an inversion of the foot. It is an abduction and never an adduction fracture. In a Pott's fracture, if the tibia fractured at all, that is, the articular surface under the internal malleolus, you have a fracture of the outer half, not the inner half, of the tibia. I never saw a fracture like this before. How high was he on the ladder?

The Intern: Only six feet from the ground.

Dr. Murphy: The patient has been in the hospital how long?

The Intern: One week.

Dr. Murphy: We have kept him here until he got over his alcoholic intoxication from which he was suffering on admission. The fibula is intact. A little more than half of the anterior part of the articular end of the tibia was fractured, and the shaft of the tibia was dislocated backward on the astragalus. How are we going to reduce this fracture and keep it reduced? In breaking the tibia he carried off all the front portion of the articular end clear back to the middle line where the astragalus slopes down. The two fragments, anterior and posterior, slip downward on the convex surface of the astragalus. One fragment goes forward, the other backward. I am going to try to put in a screw or a plate to hold that anterior fragment in position, otherwise I must say to this man at the end of this operation that he is almost sure to have great limitation of extension, because the posterior fragment will strike his astragalus, or he will have a limitation of flexion because of the faulty union of the separated parts of the tibia. I told him that he was going to have a bad foot in all probability; that we were going to do the best we could in a very bad situation; that if he wanted another doctor, now was the time to select him because of my promising him a bad result. That is the proposition. The screws in a plate may hold that fragment in position for a time and then the bone around the screws may soften. It is cancellated tissue. This is the most unpleasant condition I

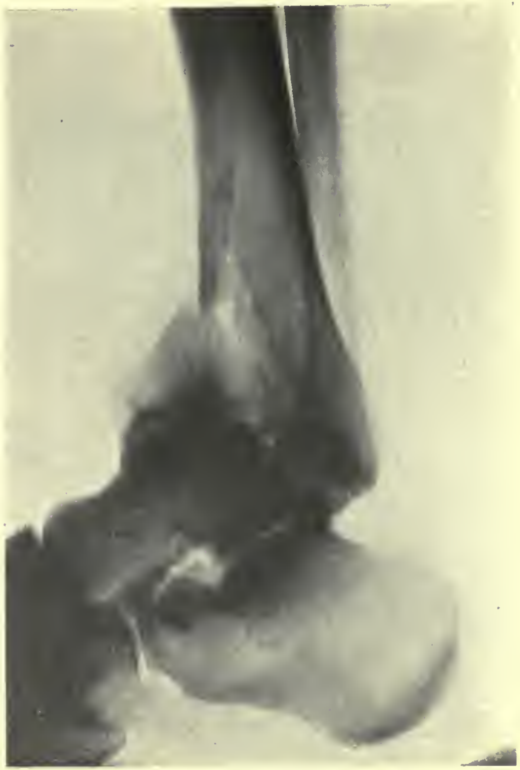

Fig. 145.—Fracture of lower end of tibia. Skiagram, lateral view, showing fracture of anterior three-fifths of articular end of tibia, with some forward displacement of the fragment and marked backward displacement of the main portion of the tibia.

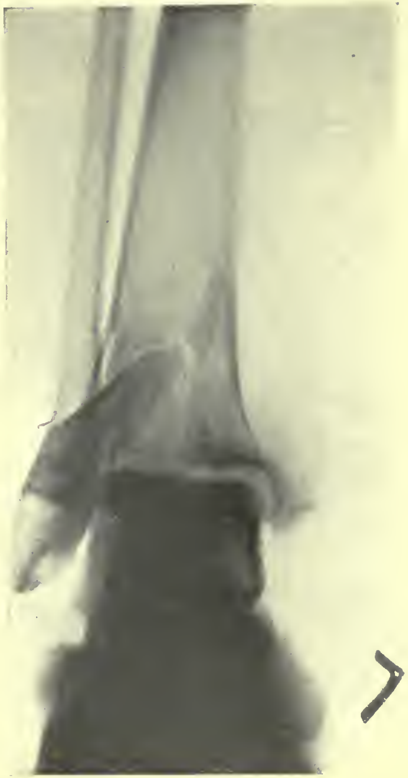

Fig. 146.—Skiagram, front view, showing displacement of the tibia outward and backward, behind lower end of fibula. The fragment appears to be continuous with the shaft of the bone.

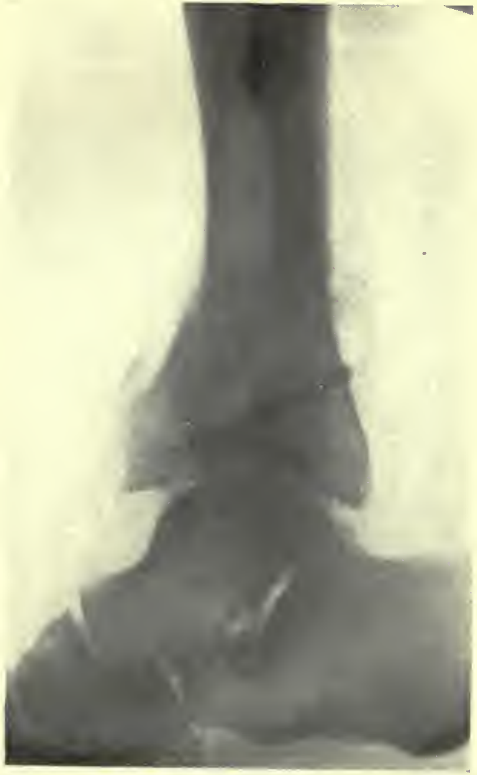

Fig. 147.—Skiagram, lateral view, showing reposition and fixation of fragment with a screw. The normal anatomy of the articular surface of the tibia has been restored. Union has taken place.

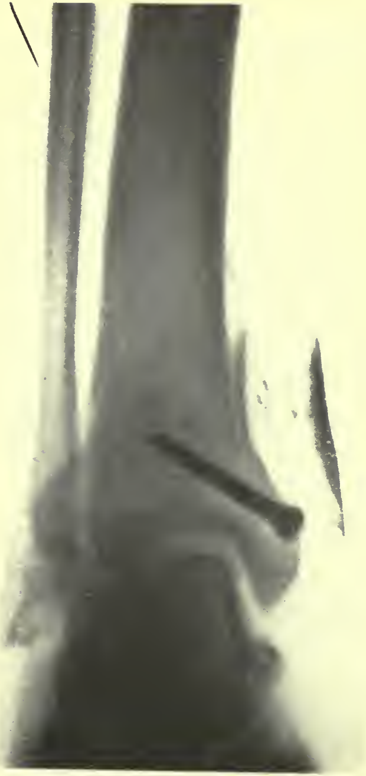

Fig. 148.—Skiagram, anteroposterior view, showing how screw was inserted from inner and anterior side of fragment, passing outward and backward. Line of fracture has been obliterated almost entirely by firm bony union.

have ever encountered in this line of fracture. Where the posterior portion of the bone is taken off we can put the limb in extreme flexion and we can hold it there and obtain a fair result.

The moment we open that ankle we are making a compound fracture of the ankle-joint. In Billroth's Surgical Pathology for 1876 the statement is made that all compound fractures of the ankle-joint should be treated by immediate amputation. Why is that statement made? Because when these cases become septic, as they did in that time, a large percentage of the patients die. The conditions for fatality now are just as great as they were then—if infection occurs. That is the point I want to bring out, and we will have to exercise extreme aseptic precaution in dealing with this case.

The skiagram shows that the fibula is not broken, and I think that is right, so far as I am able to make out. It does not get out of position. That is one advantage. The next thing I want to do is to take the posterior portion of the tibia that is luxated backward and bring it up into position in order to approximate it to the anterior portion and then hold it there with an ordinary metal screw. By making gentle but firm and steady traction on the foot we can secure this apposition.

We need not open up the ankle-joint at all. We will merely make an incision over the inner malleolus so that we can insert the screw into the fragments. This incision need not be more than two or three inches long, just enough to give us easy access to the fragment. We must be careful not to open the ankle-joint. The small fragment is at once brought into view. It is freely movable and is easily contacted with the shaft of the tibia.

We now have these fragments in accurate apposition. See how perfectly the lower fragment fits into the upper. I must secure that approximation with a screw from high up and in front to try and hold that lower fragment in apposition with the upper. If necessary, I can put in more than one screw, which will be more effective than putting on a plate in holding these fragments firmly together so that they will unite. I hope I can keep the lower fragment from sliding down on the astragalus, so that the one fragment cannot slip by the other one.

In the management of fractures of this type the question arises whether we will treat them by the open or the closed method, and it is a very debatable one at this time. Remember, I do not mean specifically this fracture, because you can see how rare a fracture this must be when I tell you that I have never seen one before; but I am speaking now of fractures in the neighborhood of the ankle-joint that are of the simple type. It is my conviction that they should be treated as closed fractures when they are in close relationship to the joint—namely, that the joint should not be opened. Furthermore, in treating fractures of the femur and fractures of the tibia by the open method we have to have a definite time for operating them, that is, eight to ten or twelve days after the accident, and never immediately after the accident. By that time the tissue reaction to the irritation caused by the trauma has developed and you have a coffer-damming of the lymph-spaces so that everything is in fairly good condition so far as resistance is concerned. The next question is, if you treat cases eight or ten days after the injury, what is the likelihood of infection? That is difficult to say.

Mr. Lane, of London, rarely ever has an infection, according to his own statement, and I know his statements are absolutely reliable. But if one sees him work, one cannot help being impressed with the exactness of his technic. He never lets anything touch the field of operation but the instruments. The same is true with us. We do not put our hands near the field of operation. When we use sponges they go directly into the field, a fresh sponge being used each time, and the sponge must not have come in contact with anything but the wound. That is the key to success in this work. Lane can do more effective work with fewer instruments in working on bones than any man I ever saw. You can see why a man so expert in his technic can have such good results. One is also impressed with the fact that a man must be possessed of that expertness in order to get the results, because the results of the open treatment of fractures in this country, as was demonstrated by Dr. Davison at a recent meeting of the Chicago Surgical Society at the Cook County

Hospital, are not very flattering. If I remember correctly, he showed six or seven cases of suppuration and non-union, the sequence of the open treatment. That is not the usual sequence of the open treatment. It is the sequence of the open treatment in hands that are not trained to do the work with the expertness that Mr. Lane has.

The lower fragment in this case swings in all directions, showing how perfectly movable it is. I now have perfect apposition of these fragments. Holding them firmly in place, the whole leg moves as one piece. If I have succeeded in keeping out a sufficient number of infective microörganisms, we will have a splendid result—much better than I anticipated in this case. The leg now is as perfect as anything can be. In doing bone work and joint surgery one must be more exacting and careful than in doing abdominal work. The abdominal tissues have very much more resistance than bone has. True, if you fail in your abdominal work you have a quicker fatality, but if you fail in this kind of work you have a bad record that is perambulating—one that causes you very much annoyance. However, that is not the important part. The important part of it is the evil result to the patient. Unfortunately, in doing much of this work one has to treat a class of people who are overfed and underworked; some of them are superintoxicated, and their resistance is much lowered. We know that when such people have pneumonia and other infections they have very little chance to recover.

I will secure these fragments with a screw. Unfortunately, all the screws made for this work are made on the artistic plan. What we want is an ordinary plain flat-head screw that has a sufficient groove in the head so that we can insert a screwdriver well into it. We must be very careful in inserting this screw so as not to break the bone. I will put in this screw on the anterior surface of the anterior fragment, from below upward. It goes in very well. The deep tissues are sutured with catgut and the skin with horsehair. The usual bismuth subiodid dusting-powder is used and collodion soaked gauze. *No drain.* We are applying now a 5 per cent. carbolic gauze. It is the old-fashioned antiseptic gauze, and the reason for using this in connection with all this bone and joint work is that there is a certain amount of oozing. This gauze will take that up and prevent it from decomposing. That is what the old-fashioned 5 per cent. carbolic gauze does. If you use the ordinary sterile gauze, it will have some odor after a few days, due to the saprophytic decomposition.

A plaster-of-Paris cast will be applied, with the foot at right angles to the leg and inverted. The cast will include the foot—not the toes—and extend well up on the leg. It will be cut in front as soon as it becomes hard, a procedure which is a routine with us in every case where a cast is applied. That will allow of spreading, and when the tissues swell, there will be no danger of a myositis occurring or any other of those disagreeable consequences of a tight cast. The cast is cut with a Gigli saw, which is placed in position before the cast is applied.

[The stitches were removed on the tenth day. The wound healed *per primam*, and the result was all that could be desired. The ankle-joint was not stiff. The tibia was firm. Three months after the operation motion in the ankle-joint was nearly normal. Firm bony union had taken place.—Ed.]

UNUNITED FRACTURE OF HUMERUS

HISTORY

JANUARY 20, 1912, while working as a laborer at a Michigan lumber camp, patient was struck on the right arm by the branch of a falling tree, receiving a comminuted compound fracture of the middle third of the right humerus. So far as could be ascertained from patient, who speaks little English, and no interpreter being available, the wound discharged for three weeks, there was no open operation performed, and no pieces of bone were removed. Reduction of fracture was attempted by manipulation and the arm dressed in wooden splints. After a period of three months examination revealed absence of bony union.

The first operation in this case was performed by Dr. Murphy in April, 1912. An anterolateral incision between the deltoid and biceps was made, exposing the fragments of the humerus. The end of the upper fragment was adherent to the skin and was freed from surrounding tissues with a periosteotome, so as not to cause injury to vessels and musculospiral nerve. This gave a full exposure of the bone. A band of tissue between the fragments, preventing contact of the bone-ends, was cut. Although the skiagram showed that there was apparent contacting of the fragments, this was found not to be the case when they were exposed. The lower fragment was next freed. A sequestrum that had united to the lower fragment was not removed. The medulla of the upper and lower fragments was reamed out for one inch preparatory to inserting the transplant.

The transplant, five inches long by 3/8 by 5/8 inch, was taken from the right tibia with the usual precautions and pointed at the ends, so as to fit accurately the cavities previously prepared in the upper and lower fragments of the humerus. The transplant was first inserted and driven into the upper fragment and then into the lower fragment, and fixed with phosphor-bronze wire loops below, to give additional strength and support. The sequestrum was used as a splint for the transplant. The periosteum was removed from the transplant. Catgut sutures united the muscles and fascia. The skin was closed with horsehair. Bismuth subiodid powder. Collodion dressing. No drain. Forearm was flexed on the arm with hand on body and a plaster-of-Paris cast applied. The wound over the right tibia was closed with catgut and horsehair and dressed, with the exception of a plaster cast. Healing *per primam* in both.

Patient returns now, four months after operation, because of lack of union of the fragments. He says he has been at work in a laundry and polishing woodwork and metal for over three months, constantly using the hand of the affected arm. He has worn a plaster cast all the time, but because of the use of his hand—and arm to some extent—it is not giving him the support which it should and would have given if the arm had been kept at perfect rest.

COMMENTS AND OPERATION

This is the first failure of union in a case where we transplanted bone for non-union of a fracture. The skiagram shows that the transplant is still in place. It seems to be firmly united to the upper fragment, but the lower fragment has freed itself from the transplant. It was implanted in the medulla, and on account of something which the patient did, probably his work, it has luxated out of the medulla and rests transversely against the lower fragment. The transplant was placed in there April 25, 1912. Whether he broke the fragments loose through an injury or not I do not know. The man is a Russian, and he gives no history of having fallen after the operation, but something occurred that threw the transplant out of the medulla. We have an opportunity today, however, of seeing the bone that has been transplanted four months ago. We also had such an opportunity in one of Dr. Golden's cases, a case of osteitis fibrosa cystica of the tibia, in which he transplanted seven and one-half inches of bone. There was a slight mild infection at the lower

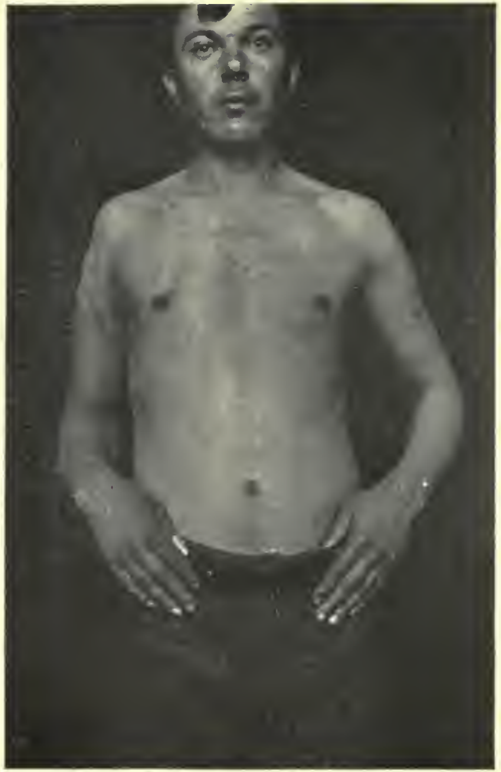

Fig. 149.—Fracture of right humerus. Photograph, anterior view, showing position of arm as supported by thumb hooked in belt. The site of the fracture is marked by a crease in the skin of the arm on a level with the nipple.

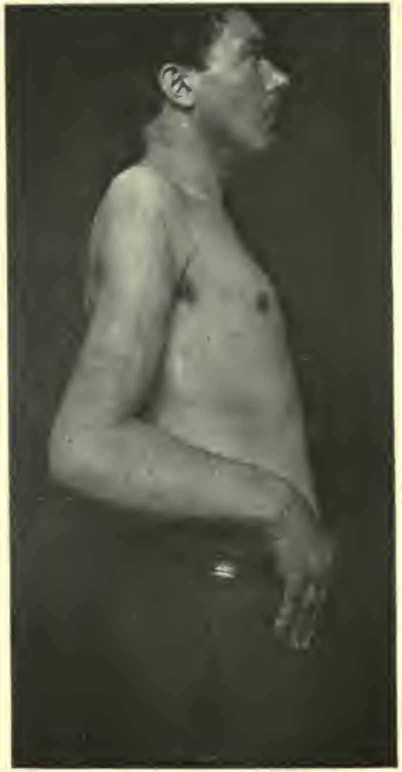

Fig. 150.—Lateral view, showing the upper end of the lower fragment projecting forward under the skin of the arm, on a level with the nipple.

Fig. 151.—Skiagram, anteroposterior view, showing line of fracture but absence of deformity. There is apparently a perfect alinement of the fragments.

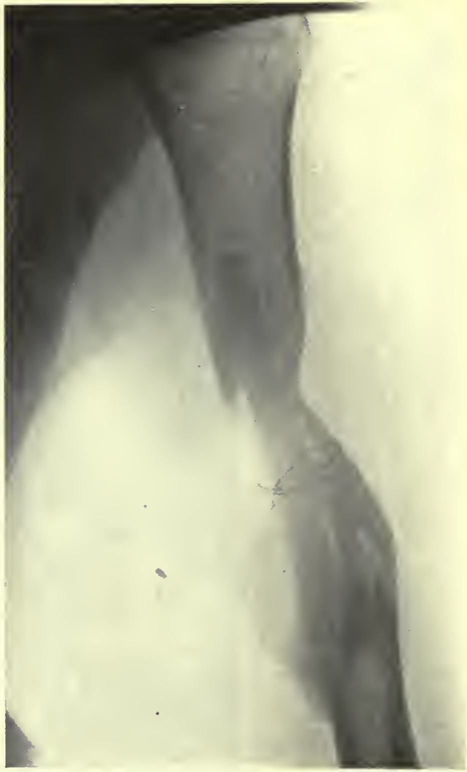

Fig. 152.—Skiagram taken after operation. The transplant is inserted into the medulla of the upper fragment and held in firm apposition with the lower and intermediate fragments by means of two phosphor-bronze wire ligatures. There is considerable callus formation around the lower end of the transplant.

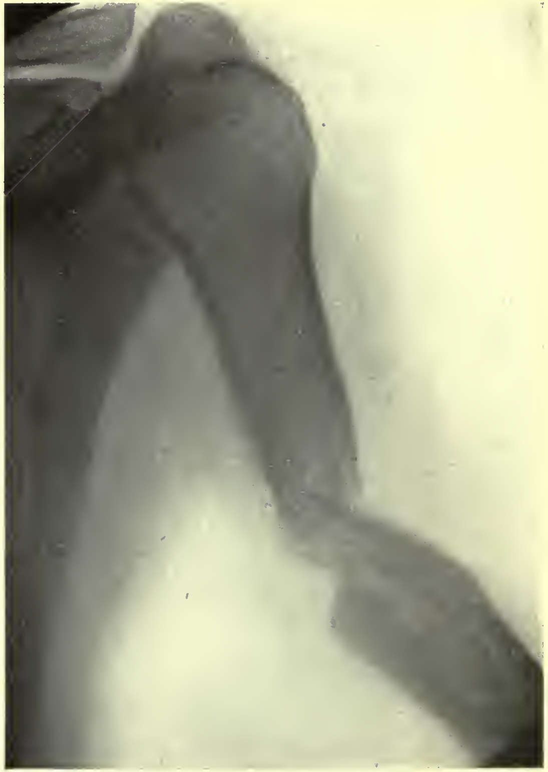

Fig. 153.—Skiagram from behind, showing the two main fragments and a small detached fragment of bone between the ends of the larger fragments. The deformity of the arm is apparent.

end of the transplant. The upper end healed in perfectly. The patient was examined some months after the transplantation. The transplant was intensely vascular, and of the same size and shape as when it was originally inserted. We implanted another small piece into the transplant, and complete union has now taken place.

We will now cut down again on this bone and see what has occurred. The upper fragment was sticking into the skin at the time we operated. There was the interposition of fibrous tissue which prevented primary union of the fragments.

We will make an incision on the outer side of the arm next to the scar of the first incision, and then excise this cicatricial tissue. The old fracture is now exposed. The point of non-union is at the lower fragment, as the skiagram shows. There is granulation tissue formation. We see plainly the sequestrum which we permitted to remain at the primary operation. There is the encircling phosphor-bronze wire that was supposed to hold and did hold. The wire did not break, but it appears to have cut off a segment of the fragment. The encircling wire seems to have divided the fragment of bone. I will free these fragments from the surrounding tissues and then decide what I will do. We may have to make another transplantation. I have the transplant in view. It is of the same size and shape as when it was put in originally. It is alive, full of blood-vessels, and bleeds as normal bone. It is firmly united to the upper fragment, but it is not united at all to the lower fragment. I am glad to see this because it is something we have not had an opportunity of seeing before. We must get the granulation tissue out of this position and prepare the bone for another approximation.

We have enough bone here in the lower fragment to make a good lateral splint for the transplant, and we can do this without taking another transplant from some other portion of the body. The transplant gets all its circulation from the upper fragment. See how perfectly that transplant has become a part of the upper fragment, and how imperfectly it has not become a part of the lower.

I am getting this tissue all freed. I am going to place the lower end of the transplant back into the upper end of the lower fragment and fix it in place so that it cannot be displaced again. If we can immobilize it, we will get union first of the soft parts, and finally ossific union between the transplant and the bone. I shall not remove the transplant, as it is securely united to the upper fragment.

The question of what happens to a transplant is beautifully shown in this case. If there be contacting and immobilization, the result is a complete vascularization, and finally complete and firm bony union. That is the purpose of the interosseous splint, and that is the purpose for which the transplant is put in. Here, evidently through some accident, it broke loose from the lower fragment. I do not know what it was. If the bony surfaces do not contact, and if the transplant is loose in its bed, union cannot take place. That is demonstrated conclusively in this case. This man has been working since the operation was performed. He had a complete primary union. He had all the conditions, as the skiagram showed, that were favorable, with the exception of the luxation.

We do all this work without a constrictor on the arm in this particular position. A constrictor could not be put on high up, and if you use one, lower down, there is danger of constricting the musculospiral nerve; besides, the bleeding amounts to little or nothing, except the annoyance that it gives one in doing the work.

I will immobilize the transplant in its bed in the lower fragment by means of screws. First we will make three drill-holes through the outer wall of the lower fragment. Through the lower two holes we will insert two screws which will impinge against the distal end of the transplant and crowd it over against the further wall of the medullary canal. A third screw is passed through the upper drill-hole and will ride on top of the fragment, so as to hold it down in its bed. These three screws will firmly immobilize the transplant so that bony union can take place. The screws will not need to be removed. They will remain in the bone—unless they become loosened because of absorption of bone around them. In that case they will be removed through a small incision.

The closure of the wound will be made with deep catgut sutures and horsehair skin sutures. The wound will be dusted with bismuth subiodid powder and sealed with collodion gauze. *No drain.* We will put on a plaster-of-Paris cast and immobilize the arm a longer time than usual. We believe we will have bony union now, because we have fixed the transplant to the distal fragment in a firm position.

The fate of the transplant is this: First, following the implantation of the bone, a blood-clot forms, surrounding it. Next that blood-clot becomes organized and vascularized. Then the vessels from the neighboring structures surround the transplant; finally, the vessels from the neighboring bone coming from the Haversian canals and the canaliculi, pass into the Haversian canals and canaliculi of the transplant, and it is these Haversian vessels that carry with them the osteogenetic elements that produce the new bone. When the Haversian vessels in the neighborhood pass into the transplant, they first produce absorption of the margin of the canal, then they cause a deposit of bone on the vessel. Finally the Haversian canals, canaliculi, and lacunæ are filled with new vessels, and these new vessels carry the osteoblasts which produce the new bone.

We must immobilize this arm completely, as the screws do not go through the transplant. The screws are put in to maintain contacting of the transplant with the bone. They are against the side and on top of, not through, the transplant, because I was afraid of fragmenting it if I passed the screws into it. You see why I did it in that particular way. As it is I have one screw passing over the transplant and two screws impinging against its side, holding it firmly in contact with the bone. That does just as well as if I had put the screws through the transplant, without the danger of injuring the transplant.

Let the record show that the transplant was organically and firmly united to the upper fragment; that the transplant was completely vascularized and bled from its surface; that it still retains its exact original shape; that it had freed itself from its implantation in the lower fragment, and it had come out of the medulla; that the lower fragment now surrounds the transplant. I fixed the transplant to the lower fragment by means of three screws, none of them penetrating the transplant, but securing the transplant in contact with the inner margin of the medullary canal. [The cast was removed four weeks after the operation, and primary union was found to have taken place. The stitches were removed and a fresh cast was applied.—Ed.]

TENOPLASTY FOR OBSTETRIC PALSY

HISTORY

PATIENT, a married woman, aged twenty-four years, came to Mercy Hospital on account of contraction of the inner and outer hamstring muscles and loss of power of extensors of foot. The limb was normal previous to her confinement, November 26, 1911. She was in labor for a long time, and then had a tedious and difficult delivery with forceps. Was under chloroform anesthesia for two hours. External genitals were bruised and perineum lacerated in the delivery. When she awoke her left leg was cold, numb, and powerless. For six weeks it lay limp in bed, without sensation or motion. At the end of this time sensation and muscular power began to return—first in the hip. She gradually recovered power of flexion, adduction, and abduction of thigh. As the power was returning in her muscles her leg became flexed on her thigh. At the end of January, 1912, she went to a hospital to have her leg straightened, as her knee was flexed to more than a right angle. A Buck's extension was put on, but this did not improve her condition any, and after two weeks she was anesthetized and the leg manipulated. This procedure succeeded in straightening the leg.

After this forcible extension a plaster-of-Paris cast was put on for one week, and then a Buck's extension for another four weeks. At the end of this period of time the limb was not straight. Since extension was taken off it has become flexed again, but not so badly as before. She has to get about on crutches now. At present she has full return of sensation and full power in the muscles supplied by the anterior crural, obturator, and internal popliteal nerves. Her foot is in a condition of equinovarus, owing to the paralysis of the external popliteal. Faradic current, active response in all nerves of limb except internal popliteal; this gives no response except an occasional sluggish contraction of peroneus with a very strong current. Anterior tibial group gives no response whatever. Has no trouble with her bowels; full control over bladder and rectum. Heart and lungs normal.

COMMENTS AND OPERATION

DR. MURPHY: We have to make a diagnosis from the history in this case. What kind of motion did she have in her hip?

THE INTERN: She could abduct the leg, and she could flex her thigh on the abdomen.

DR. MURPHY: What is the matter with her?

THE INTERN: She has had a traumatic injury of the lower portion of the sacral plexus.

DR. MURPHY: What do you mean by that?

THE INTERN: The first, second, and third sacral nerves were traumatized.

DR. MURPHY: What makes you think so?

THE INTERN: Because of the return of function in the muscles supplied by these nerves.

DR. MURPHY: No, you are in error about that. She had paralysis first of all muscles, did she not?

THE INTERN: Yes, doctor.

DR. MURPHY: Of her what?

THE INTERN: Of the lumbosacral plexus.

DR. MURPHY: What group was first involved? She was unable to do what?

THE INTERN: She had no power in the muscles.

DR. MURPHY: She had what kind of paralysis? A flaccid paralysis, did she not?

THE INTERN: Yes, doctor.

DR. MURPHY: Not a tonic paralysis?

THE INTERN: No, doctor.

DR. MURPHY: What does that mean, first?

THE INTERN: That it is a lesion of the lower motor neuron.

DR. MURPHY: She had paralysis of what else?

THE INTERN: Of motion and sensation.

Dr. Murphy: If she had paralysis of her upper motor neuron resulting from an embolism in connection with her confinement, what kind of paralysis would she have had?

The Intern: A tonic contracture in place of a flaccid condition.

Dr. Murphy: Where was the lesion first? Was it of the upper neuron or of the lower motor neuron? It was not an upper motor neuron paralysis. Why? Because it was a flaccid paralysis.

The next proposition is, Was it a spinal cord paralysis or was it a nerve-trunk paralysis? If it were a spinal cord paralysis coming on suddenly in that way from an embolus, you would have what? You would have paralysis only of sensation or motion, and not of both combined. She had paralysis of both motion and sensation. She had flaccid paralysis of motion and a complete paralysis of sensation. What is the matter with her? What happened to her?

The Intern: The sacral plexus was stretched.

Dr. Murphy: What nerves were involved and how and why?

The Intern: The anterior crural was involved, the internal and external popliteal, and the obturator.

Dr. Murphy: Where does the anterior crural come from?

The Intern: From the second, third, and fourth lumbar.

Dr. Murphy: Yes, all right, sir. The muscles supplied by this nerve were paralyzed. Why? Because she could not flex her thigh on the abdomen. Proposition number one. Next!

The Intern: The internal popliteal was paralyzed.

Dr. Murphy: It comes from the fourth and fifth lumbar and the first, second, and third sacral nerves. It is a part of what plexus?

The Intern: The sacral plexus.

Dr. Murphy: Yes. What portion of that goes to make up the external popliteal nerve and what portion the internal popliteal nerve?

The Intern: The anterior divisions of the nerves of the fourth and fifth lumbar and the first, second, and third sacral nerves make up the internal popliteal nerve, and the posterior divisions of the same nerves make up the external popliteal nerve.

Dr. Murphy: Where does the greater portion of the nerve-supply come from?

The Intern: High up in the cord.

Dr. Murphy: If the spinal nerves you mentioned are the source of the sacral plexus, which portion supplies the external popliteal and which portion the internal popliteal? Remember that both receive branches from there, but the major portion of one nerve comes from one side above, the major portion of the other comes from below. The internal popliteal is from the lower one; the external popliteal from the upper. The obtura-

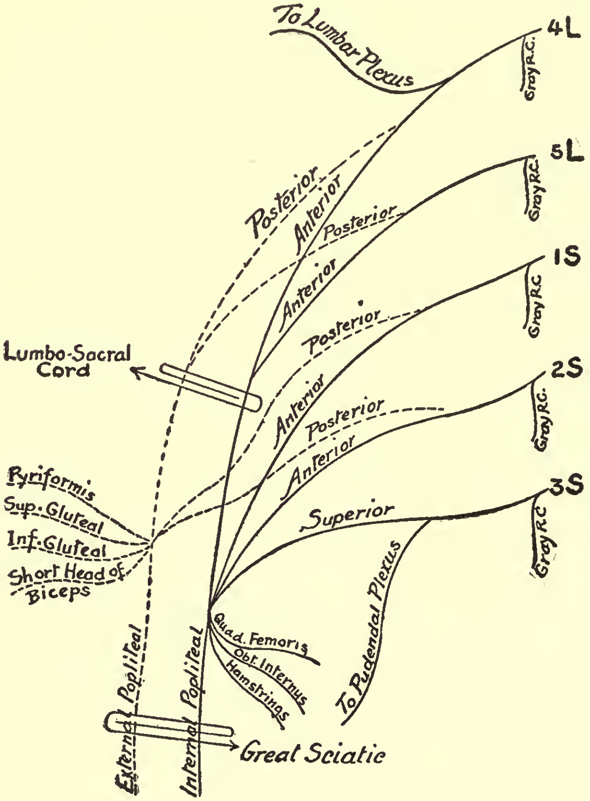

Fig. 154.—Plan of the sacral plexus.

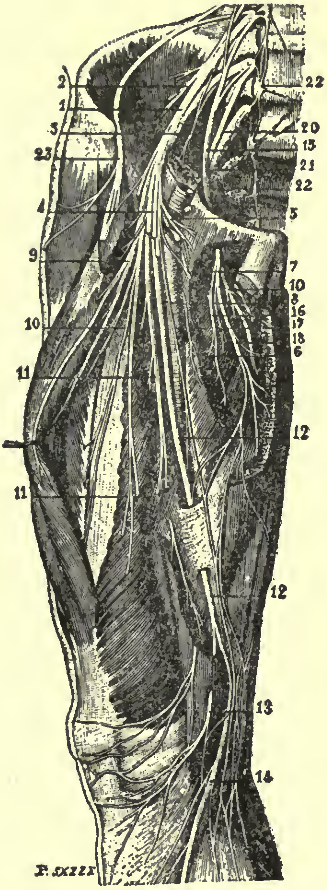

Fig. 155.—1, Anterior crural nerve; 2, 3, branches to iliac and psoas muscles; 4, external cutaneous nerve; 5, 6, 7, musculocutaneous branches; 8, anastomotic branch of internal cutaneous; 9, 10, 11, branches to rectus femoris, vastus externus, and vastus internus; 12, internal saphenous nerve, with 13, its patellar branch and 14, its tibial branch; 15, obturator nerve; 16, branch to adductor longus; 17, branch to adductor brevis; 18, branch to gracilis; 19, branch to adductor magnus; 20, lumbosacral cord; 21, junction of cord and first sacral nerve; 22, sympathetic; 23, external cutaneous nerve (after Sappey).

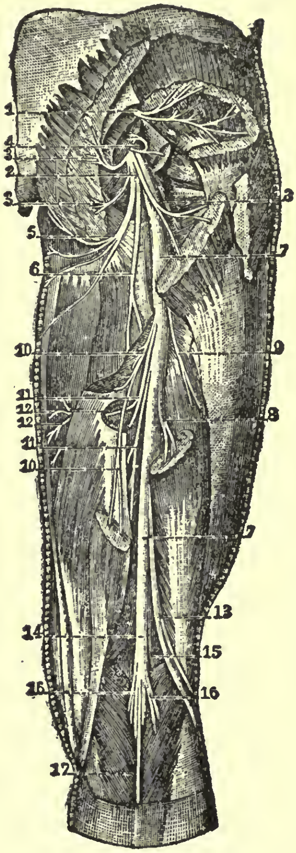

Fig. 156.—1, Superior gluteal nerve; 2, small sciatic nerve; 3, branches to gluteus maximus; 4, branch to pyriformis; 5, genital branch of small sciatic; 6, femoropopliteal branch; 7, great sciatic nerve; 8, branch to long head of biceps; 9, branch to short head of biceps; 10, branches to semitendinosus; 11, branches to semimembranosus; 12, branches to adductor magnus; 13, external popliteal; 14, internal popliteal; 15, branch to plantaris; 16, branches to gastrocnemius; 17, origin of external saphenous (after Sappey).

tor nerve comes from the second, third, and fourth lumbar nerves.

The anterior crural supplies all the muscles on the front of the thigh. This was paralyzed. The external popliteal arises from the fourth and fifth lumbar and first and second sacral nerves. Through the anterior tibial nerve it supplies the tibialis anticus, extensor longus digitorum, peroneus tertius, extensor proprius hallucis, and through the musculocutaneous branch it supplies the peroneus longus and brevis. The internal popliteal arises from the same source as the external. It supplies the gastrocnemii, plantaris, soleus, and popliteus muscles. Before the great sciatic nerve divides into the two popliteal nerves it sends branches to the flexors of the leg, the biceps, semitendinosus, semimembranosus, and the adductor magnus. All these muscles get their supply from the lower half of that sacral plexus, that is, their major supply.

Let us see what is going on in this case and why. We have what? We have a return of motion in the flexors of the thigh supplied by the anterior crural nerve. We have a return of motion in all the muscles supplied by the internal popliteal. That means what? It means that you have the gastrocnemius and soleus, the external and internal hamstring groups, all having restoration of function. We have left what? The one nerve in which there is no restoration of function is the obturator, the nerve that arises from the second, third, and fourth lumbar nerves, and that supplies the obturator externus and the adductor brevis and longus.

When the head of the child is two hours and a half in coming through the lower strait with forceps applied and probably pressing on the nerve, you can see that it is subjected to greater pressure than any other nerve. So much for the anatomy, which is exactly in consonance with the findings. They are typical and clean-cut.

The question arises, What is going to happen to these nerves. We can say now with certainty that the axons of the internal popliteal group and of the anterior crural group are all going to be fully restored. It is nine months since this woman was confined. What is going to happen to this one trunk that rested there and had the forceps on the child's head for a considerable period of time pressing on it, and why? This brings us back to the traumas of nerves. When the trunk of a cerebrospinal nerve is divided and the ends are brought in contact, no matter how long after the division, so long as there is axonal contact you have always a regeneration, except in the nerves of special sense. Remember, the cranial group of motor nerves and the spinal group of motor nerves all originate from a continuation of the motor tract. If you go from the spinal cord up into the lower brain, it is the same tract exactly; or, if you turn it around and go from above downward,—and I am speaking now of the lower motor neurons,—all these originate from the same tract. When you come to the sensory nerves, you have exactly the same condition. The sensory nerves of the head group or of the cranial group have one great ganglion, the Gasserian ganglion, which is analogous to the ganglion on the posterior root, except that in the head it is situated inside the foramina, while in the spine it is situated in three different positions. First, high up, just outside, then just inside, and lower down in the caudal zone it is situated completely inside the canal. All this is so simple when you get the maps properly arranged in your mind. We have been speaking about axonal contacting—that is, after division, after incision-division. Supposing I cut a nerve with a knife, then bring the ends together, no matter how soon you reunite them, the distal portion first degenerates, and then regeneration proceeds from the proximal side. The other axons that do not regenerate are the axons in the spinal cord itself. You ask yourselves the question, When a nerve is cut, will that nerve regenerate?

It would be difficult to remember all the nerves if the question were asked individually. Fortunately, you can group all of them, and the nerves that regenerate are those that have a neurilemma. An axon that has no neurilemma is incapable of regeneration. Therefore, when a nerve is divided the question which you ask is, Has it a neurilemma? The axons that have no neurilemma are the axons in the upper spinal cord, not in the cauda. The axons that have neurilemma are the cerebrospinal nerves, except the nerves of special sense—the acoustic, the gustatory, etc. Therefore, you have two sets of axons which are absolutely and always incapable of regeneration when divided—those in the spinal cord above the cauda and the nerves of special sense. We all know that when the optic nerve is cut or is degenerated, it never regenerates. Why? Because its axons have no neurilemma. We all know that when any of the spinal nerves are divided and brought in apposition, with axonal contacting, you always have a regeneration.

There is another type of trauma which has a much more unfavorable prognosis than in the case of those nerves after division. I refer to the contusions and the stretchings of nerves. For instance, take a man who has his arm caught in an engine shaft, is swung around the shaft several times, and he comes out with his arm paralyzed. You commence to dissect out his brachial plexus from the spinal cord, and you find no division of that nerve at all; still he has a paralysis that is permanent. I remember a man who was standing near a boiler that exploded and caught him here [referring to the shoulder], threw his shoulder out, stretched his brachial plexus, and paralyzed his arm. We exposed the brachial plexus over its entire extent; there was not a single division anywhere in it; but when we took the strands of that brachial plexus in our hands and slipped our fingers along it, there was a rosary of deposits of connective tissue between the parts of the divided axons in the nerve. That portion of the nerve was filled with newly formed connective tissue. Now, where there is a contusion of a nerve the external covering frequently is not cut at all; the internal portion is divided and there is a deposit of connective tissue in spots. Fortunately, when it is at one point, and it is a hard nodule, like a neuroma, you can take out the neuroma, approximate the cut ends of the axon, and you get a perfect functional result. But, notwithstanding that apparent approximation, without resection there never will be the slightest restoration of function until you take out the neuroma.

This brings us back to the literature of repair of the spinal group of nerves. If you will look over the literature for one hundred and twenty-five years, you will find that nerves have been divided and approximated, and you will find two distinct schools, one saying that when they are divided and united, motion returns; the other maintaining that when they are divided and united, motion does not return. Why this diversity of opinion, yet both based on clinical observations? They are not dreams, but clinical observations, and both are absolutely correct. When we analyze the situation we find that the reason for this diversity of opinion is that the contacting of the axons was not appreciated. One man was fortunate enough, when he took a divided nerve, to cut out enough of it to get axonal contacting, the same as one gets contacting of a cable wire when it is broken. The other man merely brought the ends of the nerve together, put them in contact, but the ends were covered with fibrous tissue and insulated, and there was absolutely no effort at regeneration across the line of approximation. In making nerve repair, whether after division or contusion, you must cut off the neuromatous end of the nerve until you see the bundles or fasciculi of the nerve sticking out like the bristles of a brush. Unless you go back to the position where these are demonstrable; unless you have them at both ends and get contacting, without the interposition of connective tissue, you will not have regeneration of the nerve. If you bring the divided ends in contact and protect them against infiltration from connective tissue from the side, there will be regeneration. What do I mean by that? The ordinary nerve connective tissue is a neuroglia—a different type of connective tissue entirely from that in the neighborhood. The nerve axons penetrate that neuroglia without any trouble at all. They will not penetrate ordinary fibrous connective tissue. It is very important to have the ends freshened in order that regeneration may take place.

Wherever you have a connective-tissue mass, as in the stretching or contusion of a nerve, if you expect to have regeneration after a certain time has elapsed and its axons have not contacted, there is only one thing to do, and that is to resect that hard mass and make an end-to-end contacting of the nerve. If you do that, you can have restoration of function.

In this case nine months have elapsed, and there is no response to faradic stimulation of the external popliteal, meaning that the nerve was compressed and might have in it a mass of fibrous tissue, produced from the outside, and not from the neuroglia, that may never be penetrated by the divided axons that were crushed off in the delivery of the child. But we still have to wait for another nine months. It takes about eighteen months before one can eliminate the probability, if not the possibility, of restoration of function. There is, however, a tonic contraction of the muscles and deformity of the limb. That can be fully remedied and a leather cast put on. This woman can go about her work and receive impressions from the axons of that trunk that is out of commission entirely. I believe voluntary effort is a very important matter in the restoration of function.

We have found that if a nerve has a neurilemma it is capable of regeneration. In working out the surgery of the nerves that has been the basis of our analysis. The histologists and the physiologists are still in controversy as to whether the central nerve body of a motor neuron and an axon are one and the same protoplasmic cell body, with a cell body of a single nucleus in the anterior cornu of the cord. Some say it is, others say it is not. To us as surgeons it does not make any difference. It is my belief, and that belief is based on clinical observations, that the axis-cylinder of a neuron is a continuation of the protoplasm of the central cell body, and, following a biologic law, if you cut off any portion of a cell from its nucleus, the portion distal to the division always dies. That is exactly what happens in the axons or the axis-cylinder of the neuron—if it is severed, the distal portion always dies. Instantaneous or immediate contacting has no effect whatever. It always dies, and it must regenerate from some force that is received from the proximal or central cell-body portion. The axis-cylinder or axon will not come down beyond the line of division unless there is a contacting with an axon that has a neurilemma.

That led Kennedy and others to the belief that that distal portion, the neurilemma axon in the distal portion, produced a something, not an axis-cylinder, but an embryonal axis-cylinder which follows the tract on down. I do not believe it. I believe that the same thing happens as in the case of bone and muscle, namely, that it produces a tube of the right shape and size and affinity. Chemotaxis was the word used by Toma. It produces a tube that has a certain affinity for the axon, and the axon goes right through, just as in the case of bone. If you put one end of a piece of bone, eight or nine inches long, in contact with living osteogenetic cells, the Haversian vessels of the original bone will pass into the Haversian canals and canaliculi of the transplant, and pass throughout its entire length. But they will not pass down the entire length in the case of ox-bone, cat's-bone, or dog's-bone. It must be an autotransplant. They will pass down through that bone whether it is covered with periosteum or not. There is something in the nature of an affinity or attraction that brings the Haversian vessels with the osteogenetic cells down into the transplant. There is something that takes the axis-cylinder into the distal portion of the nerve that has been divided.

In one of our cases regeneration took place twenty-six years after division. I expected, of course, that the distal portion would be small and atrophied, and that I would not be able to find it, but it was exactly of the same size as the proximal portion. It is slower in regenerating, but once the axons are contacted, regeneration will occur. The proximal portion of the axis-cylinder extended downward and there was full restoration of motion after twenty-six years. The length of time appears to cut no figure, except that the process of regeneration is a little slower.

Where is the practical application? There are two things to be borne in mind: First, to prevent the deformity of the limb. We must restore it to a straight position, and it must be kept in a straight position. We will do that by a plastic lengthening of the hamstring tendons and then putting on a plaster-of-Paris cast. Every night the cast may be taken off to keep the knee- and ankle-joints functionating. If at the end of another nine months she does not have restoration of function, we are fortunate in having the one sacral plexus nerve-trunk involved which can be reached most easily. We can go in from behind or through the peritoneum, and I prefer the latter, and expose that one trunk where it comes over the brim of the pelvis, and if necessary, resect and approximate the two ends and have restoration of function. That is the proposition as it appears to us now.

The case is a very interesting one. It is the third case of the kind that I have seen. I have seen temporary paralysis, but not a case where the paralysis lasted very long. What have we to do here for this woman? We have now a contraction of the hamstring group of muscles. We have a contraction of the extensors of the foot, namely, of the gastrocnemius and soleus, the tendo Achillis. There is a spastic contraction of these muscles which has drawn the foot in the position it is now in, and the contraction of the hamstrings has flexed her knee. If we let that continue for another nine months, her joints will become fixed, and we will not only have to elongate the tendons, but we will have to do a posterior capsulotomy, because the capsule will have become thickened and infiltrated, and, as the capsule is stronger than the bone, if you endeavor to stretch it by force, you produce a fracture, which occurs so commonly in children in connection with elbow-joint fractures. We shall have to elongate the external and internal hamstrings. The former consists exclusively of the biceps tendon. The latter is made up from the gracilis, the semimembranosus, and the semitendinosus muscles.

In the method of elongation of tendons there has been considerable experimental work done recently as to which is the better way. We found out long ago that from a practical standpoint certain tendons can be separated for an enormous distance and span a space without any of the connecting fibers having been left. They can span a space of two and a half inches. The tendo Achillis may be divided or separated two and a half inches, and if nothing is interposed between the divided ends, they will unite. You cannot do that with the tendons of the forearm, and you cannot do that with the extensors or flexors of the knee, and have union and function restored. The question arises, what material can we use to span that distance? All you need is to have some continuous contact or transmitter of the impulse of the cells for regeneration. The simplest method of elongation of tendons is the Z-shaped division. For instance, cutting through one half of the tendon, splitting it lengthwise, then cutting through the other half, a Z-shaped incision, and then bringing the long ends in contact and approximating them by suture [indicating] or spanning the distance by putting in sutures.

It would seem we have been making things more complicated of late, and if I were to make any criticisms on what I saw during my last trip abroad, particularly in the European clinics, it is this: The one thing that impressed me most was the great tendency to make things more complicated, to see how much useless or needless work could be done to accomplish a simple purpose. This was particularly striking among surgeons whose great tendency heretofore has been to simplify methods rather than to complicate them. We have finally come to the transplantation of tendons from other portions of the body, which is an accomplished fact. It can be done and done effectively if asepsis is maintained. We have come to span the space by silk, two or three or four strands of silk, passing from one end of the tendon to the other. The silk threads act as conductors of the fibrous tissues of the tendons until the ends come in contact, and regeneration of tissue occurs around the silken strands. Then there is the splitting of the tendon as I described it.

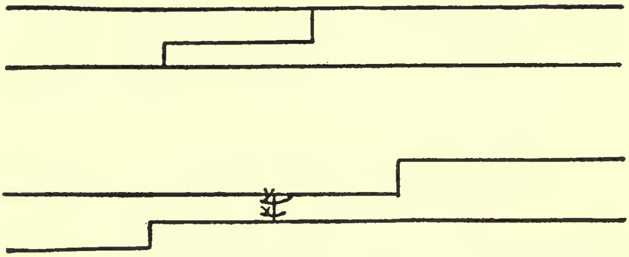

Fig. 157.—One method of tendon elongation. Double "L" incision and approximation by suture of long ends of two "L's."

In the suture of tendons, if you merely put in an overstitch, it will often pull out if the patient makes an involuntary contraction in coming out of the anesthetic. It was found by experimentation that the best type of suture was, for instance, to make a double-loop suture. That gives the greatest mechanical support. It is easy of insertion wherever you bring the cut ends of the tendon together. We do not use silk at all in our surgery, except in the intestines. We use catgut and bring about side-to-side union, and we have found it very effective. It makes a greater bulk for the time being, but it is more effective in the end. In our tenoplasties on the tendons of the forearm we always wrap the line of union. You will remember what a plumber does. When he makes a union of a piece of lead pipe he wraps it, or, as he terms it, "wipes a joint." We have employed egg-membrane for this purpose. The thing you must guard against in making tendon suture is to see to it that the ends do not cross over to other tendons and adhere to them laterally. If they do, you have the whole muscle group moving at once, instead of each muscle moving individually, although in the thigh that would not make any special difference.

In elongating the external hamstring tendon there is one thing that you must pay attention to, and I am afraid that that is not emphasized enough, and that is, to take sufficient care of the external popliteal nerve. It is so frequently divided, crushed, or overstretched, and you have as an evil result following it a drop-foot. This division of the external popliteal nerve, unless one is thinking of it, can occur in the most thoughtless way. I would not say careless. It is in close proximity to the tendon; it must be guarded and protected, or the patient will be left in a worse condition after the operation than she was before. The external popliteal nerve becomes paralyzed not alone from division with the tenotome, but it becomes paralyzed occasionally from infections in the neighborhood of the knee-joint; but, fortunately, nerves and arteries stand a colossal infection around them without ever having the infection penetrate the sheath and interfere with the function of the axons. Where there is sloughing in the neighborhood of a nerve, the nerve having less

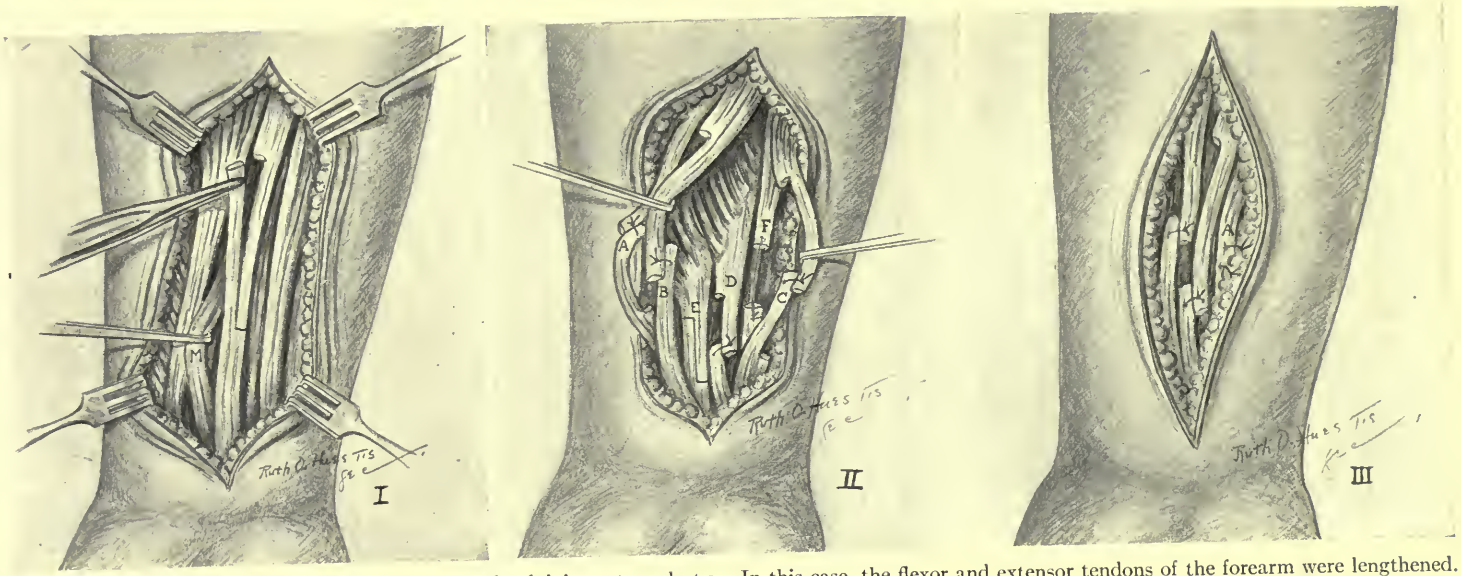

Fig. 158.—An arm case, illustrating the methods of doing a tenoplasty. In this case, the flexor and extensor tendons of the forearm were lengthened. I, Shows method of making the incision, a double ┐_. II, Shows method of lengthening the tendons (B, D, F). III, Shows how fat and fascia are sutured over the union (A) to prevent adhesion of tendon to skin.

vulnerable tissue to become involved, is less apt to be disturbed. Therefore, in doing surgery in the neighborhood of the nerve, the nerve in all human probability has not been disturbed, and you must not disturb it with your instrumentation. It must be protected, and this has reference especially to the external popliteal nerve. The biceps is attached to the outer side of the head of the fibula, and the external popliteal nerve passes along on the inner side of the biceps tendon outward and winds around the neck of the fibula. In making the incision over the external hamstring it should never be made in the popliteal fold, but on the outer side of the tendon, otherwise it often forms a contracting scar or a keloid develops in it.

We will make a longitudinal incision over this tendon, just to the outer side, and expose it. There it is, you see. There is the biceps tendon, and there is the external popliteal nerve. It is right in the field. That is why we make our incision on the side of the tendon. One should endeavor never to make scar tissue in the popliteal fold. The tendency to connective-tissue formation occasionally results in a very irritable scar, and is detrimental to the patient's comfort. The tendon is nicely exposed. I will split the tendon half way across, then split it along its long axis upward for a distance of two and one-half inches, and then complete the other half of the transverse division. I know now that the external popliteal nerve is outside and behind. There it is shining through. With the excitor we can determine whether or not we get any response. There is a partial response only. That means what? That I may not have to do the operation which I described. I can make a suture out of the tendon tissue if I desire. That is another refinement of surgery that is entirely unnecessary. It does not make quite so good a suture as the catgut. A running catgut suture will do it. We will overlap the cut ends of this tendon so as to make a lateral anastomosis, the sutures going only half way through the tendon, so as not to cut off its nutrient supply.

That is all we need to do here. We will close the skin incision with horsehair. Horsehair is the simplest type of skin suture, and we use it very extensively in this clinic. For a long time we used the subdermal catgut suture, but we have returned to the horsehair again, as it is so easily applied. On the face we occasionally use the subdermal suture.

We have to elongate the internal hamstring tendons next. We shall have to make separate elongations of the tendons comprising the internal hamstring. Here I have to make my incision lower down, and still I will not go into the popliteal space. We are wide of the internal popliteal nerve. We will elongate these tendons in the same manner as we did the external hamstring tendon.

A gradual process of stretching of these tendons would be a long and unsatisfactory procedure. We have great hopes that the ultimate outcome of this will show that the axons of the external popliteal are not only living, but that they are regenerating and capable of transmitting impulses. Remember, in a degenerated axon that could not occur. The faradic current, when applied to a living axon, gives response, while the galvanic current gives response of the muscle regardless of the nerve-supply. Remember that we made a lateral apposition of the cut ends of these tendons. We never include the entire circumference of the end with the suture. If we do, we necrotize it. We leave half of this free. We let the reëstablishment of the new tendon take place through the living half that is not involved in the suture. The catgut we are using is the chromicized fine catgut of the ordinary type. These tendons are all free from tension. Some of them have been elongated for four and one-half inches, so that there is absolutely no traction. As the nerve is practically regenerated now, there will be very little tendency to subsequent contraction. The skin incision will be closed with horsehair.

In speaking of nerve regeneration, we have come to this stage in surgery now, that when a nerve is divided, it is just as incumbent on the surgeon to approximate the ends of that nerve in the proper way by an immediate procedure to secure subsequent restoration of function, as it is for him to adjust the ends of a fractured bone. The union of a nerve is just as uniform and regular as is the union of a bone. The reëstablishment of function in a nerve is just as uniform and regular as is the reëstablishment of function of a bone. The matter of omission in this connection in the future will be considered as serious by the profession and by the people as will be the omission to adjust the fragments of bone properly after a fracture. It is an important matter for the profession to take cognizance of this fact, for unless care is exercised in the future, we are going to get into legal complications in connection with this class of cases, just as we do in connection with fractures. That is not the serious part of it. The most serious part of it is that we incapacitate the patient, and it is too bad if we have to be instructed as to what is the proper thing to do through the courts. We must take cognizance of these advancements, and not only that, but we must apply them in practice and at the proper time.

I previously remarked that it was my observation that any period of time could elapse between the time of division of a nerve and its subsequent suture, and still regeneration would take place. Yes, that is very true, but the time of invalidism depends somewhat on the delay in the reapproximation of the nerve-ends, namely, if immediately after the division of a nerve from a contusion, laceration, or incision you unite the ends, you have a more rapid return of function, provided you are able to maintain asepsis, so that at the end of six or eight weeks in some of the cases of division of the nerve in the forearm there is a restoration of function. Restoration of function after division of the lower motor neurons occurs later.

The potency of regeneration varies in proportion to the size of the nerve-trunks, and particularly when we speak of the sensory nerves; it varies in proportion to the size of the ganglion or the ganglionic center. In the case of the large bulb in the cord, where the secondary motor neurons have their nutrient cell-bodies, we get more rapid and complete restoration. There is a better and a more speedy restoration of the upper bundle or segment of the motor neurons, namely, those that come out into the brachial plexus. The next greatest potency of regeneration is in the caudal or above the caudal zone, at the conus, because that is the next largest bundle. In the sensory nerves we have the potency of regeneration controlled by the size of the ganglion on the posterior root.

If you have a ganglion that is large, you have a more rapid, complete, and persistent regeneration. We have that accentuated to the superlative degree in the potencies of regeneration of the trifacial nerve. The trifacial nerve has, on its sensory portion, the largest ganglion in the body. The regeneration of that nerve is not equaled by any other tissue, and that accounts for the recurrences we have of neuralgias of the trifacial nerve. You can divide the trifacial nerve outside its cranial foramina, dividing all branches, or you can pull them out through the cranial foramina as far back as they will pull out,—half an inch, an inch and a half, or two inches,—and they will all regenerate. It takes from six to sixteen months to have this regeneration take place. Furthermore, you can cut the trifacial nerve, turn the end downward, approximate it, and the axons will come around and find the distal axonal portion, and it will regenerate. You can plug the foramina with screws or with iron or with wax, and the fibers will get around the plug unless it has a large flat head, which occludes the foramen, so that the nerve cannot get around. It is the same as when we fill up the foramen with a plate of paraffin.

There is enormous regenerative power in the sensory axons of the trifacial nerve. There is no such regenerative power, however, in the motor elements of that nerve. When the motor root is divided, there is a suspension of transmission to the internal pterygoids and masseters, and if you have divided the nerve on both sides, dropping of the jaw occurs. You have no idea what a surprise that is when it occurs to you for the first time. I had a case in which I had to operate on both sides. To my horror and surprise, when I got through, the patient's mouth dropped open and she could not masticate. Her temporals were still intact, and finally she would bring the jaws together when she concentrated her mind on it. But when in a state of quietude or rest, her mouth was open. It is only by these unfortunate experiences that we sometimes learn a lesson. However, I did not think of it in that light. I felt the nerve would be traumatized temporarily and union would be restored. In that I was sustained in my belief. Regeneration started in the traumatized motor axons, and it was something like twelve or fourteen months before she had complete restoration of function.

We will next lengthen the tendo Achillis, making the incision directly over the tendon, exposing and then dividing it in the same manner as we did the hamstrings for a distance of two inches. The ends are then united with catgut.

In making this division and union, I took the inner half of the tendon and united it to the outer half. Why? Because the tendency of the foot is always to pull inward, and I left the outer half, attached to the outer portion of the tuberosity of the os calcis, intact, so that the muscle contraction does not tend to pull the foot inward. Here again I made a lateral approximation. I have now the assurance of reunion, and I know that this can be done aseptically, and there is no occasion for hazarding the muscle by dividing it subcutaneously. In this one case I am putting in a double loop suture of catgut.

This limb will be put in a straight and abducted position in a plaster cast right away. We extend the cast clear up into the inguinal fold, to get the full effect of the plaster cast on the knee, and down to the toes. The foot will be everted. This cast will be left on for from four to six weeks, being taken off only at such times as it may be necessary to remove the skin stitches. That will prevent contraction of the muscles and keep the limb straight. We are convinced that the final result in this case will be all we desire.

ANKYLOSIS OF THE TEMPOROMAXILLARY JOINTS

HISTORY

Patient, a school-boy, aged fifteen, comes to hospital on account of fixation and deformity of lower jaw. When six months old a swelling developed rather suddenly in front of the tragus on the left side. A few days afterward pus discharged from the meatus on the left side for four or five days, and then the swelling in front of the ear disappeared. There was no swelling over mastoid then or since, and there has been no further trouble from that ear.

From what his parents told him he had no throat or skin affection and there was no noticeable deformity of his mandible then, and it was not until twelve months after his ear trouble that they found that his lower jaw was fixed. Since then he has been unable to open his mouth. His lower jaw is deformed in that the normal angle of jaw is absent and the symphysis menti narrow and pointed, and a little to the right of the middle line. The molar teeth are in corresponding positions on both sides, but his upper front teeth project beyond the lower about one-half inch. Skin for a thumb's breadth to the left of the symphysis menti is contracted. This contraction extends into the tissue of neck. The lower jaw is pressed firmly against the upper, and he is unable to move the jaw either way more than one-eighth of an inch. There are no enlarged glands to be felt in neck, no discharge from either ear, and no signs of old or recent mastoid trouble. His heart and lungs are normal and otherwise he is in perfect health. Urinalysis negative. Blood examination: White blood-corpuscles, 8800; red blood-corpuscles, 4,750,000; hemoglobin, 75 per cent.

COMMENTS AND OPERATION

Dr. Murphy: How old was the boy when this occurred?

The Intern: Six months old when a swelling developed in front of his left ear.

Dr. Murphy: And he had a discharge from his ear?

The Intern: Yes, doctor.

Dr. Murphy: It did not develop behind his ear, but in front of his ear. It discharged for four or five days, following which he had a gradual fixation of his lower jaw. He has had complete ankylosis of his jaw since he was seven or eight months old. The result is that his jaw is in that condition that one finds in congenital aplasias of the lower jaw, in which the lower jaw recedes an inch or an inch and a half. He has a condition resembling that which is the result of the fixation of his jaw. In the skiagram we note the direction of his jaw. There appears to be little or no angle of the lower jaw. It runs almost straight from the chin to the articulation, right straight back.

What happened? What happened to the boy when he was six months old? In all probability he had an otitis media. He probably had an infection, occurring, first, in the neighborhood of the mastoid, where it makes its way to the surface. Next, in front, at the attic, and lastly, coming through in front. The boy had practically no ossification in these bones at that time, and the infection came through in front and in all probability went into and involved the articulation of his lower jaw. This is our belief about this type of infection. It is, however, to my mind, only one of the things that occur to produce the effect which we have in this case.

I had three such cases of infection in my office this week. One of the patients had a suppuration. The suppuration burrowed through the tissues of the cheek and made the chin swell. That case I believe may have had an ankylosis from infection, but I believe that that child's case was a fibrous contraction of the cicatricial tissue that developed from infection in there.

Any one doing mouth surgery knows how these internal

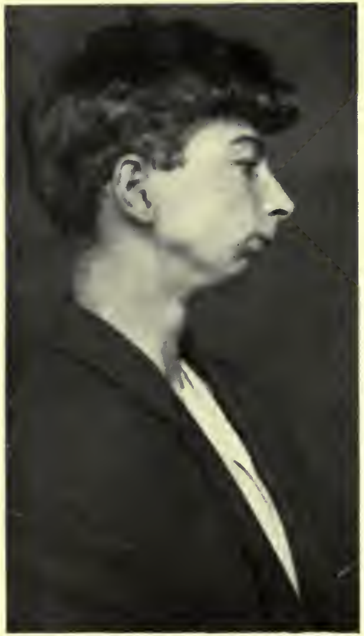

Fig. 159.—Ankylosis of jaw. Photograph taken before operation, showing extent to which patient could open his mouth. The bony ankylosis was in the temporomaxillary joint.

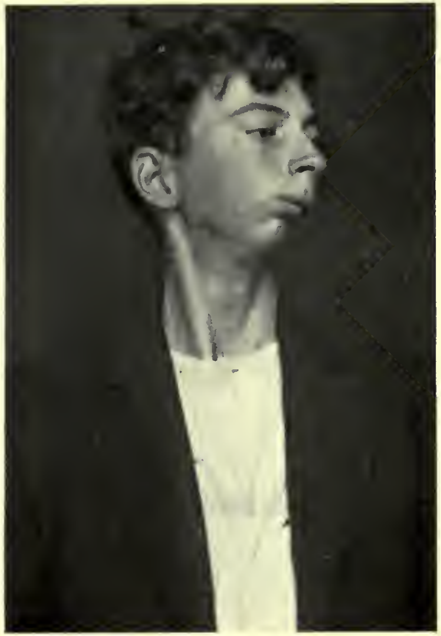

Fig. 160.—Photograph showing marked deformity of chin, due to the fixation of the jaw.

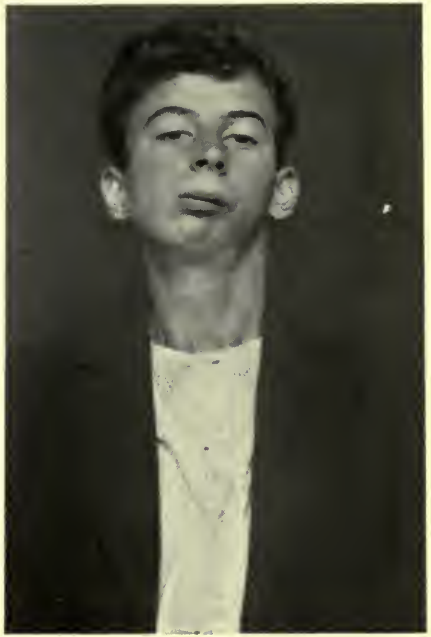

Fig. 161.—Full-face view, showing the chin drawn over to the right side, with considerable flattening of the left half of the face.

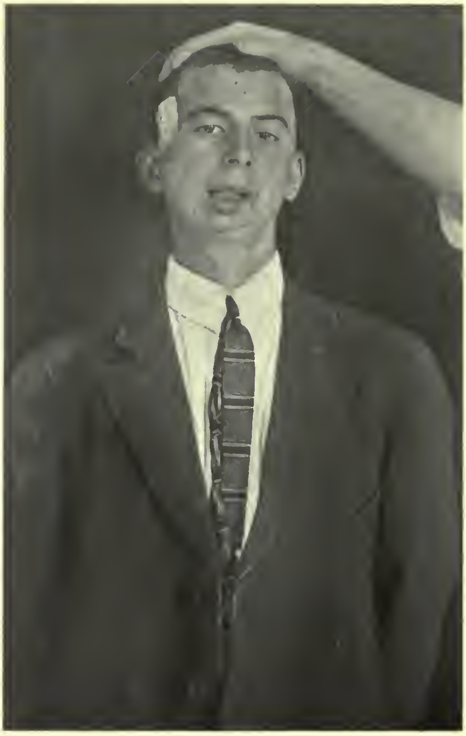

Fig. 162.—Photograph taken after operation and showing extent to which mouth could be opened before operation. A glass tube could just be passed between the teeth.

fibrous bands can bind that jaw like an iron band, and you cannot move it a particle. You can see, therefore, that in the management of this case we will have two distinct elements to consider.

This condition has existed almost since this boy was born, and he is now fifteen years old. It has been there fourteen and one-half years. If he has a bony ankylosis, we not only have to relieve it, but we must implant in between these two bony portions of the joint the temporal fascia and the temporal muscle, an operation which was originally done by Nicoladoni, except that he used only muscle. Then, when we have the bone fully freed from this connective-tissue mass, we have real trouble. If that involves the masseter muscle which is attached to the lower margin of the zygomatic arch, we have to divide it either here or where it is attached to the malar process and zygoma.

The osteal type of ankylosis without the fascial contraction, and particularly where the ankylosis has been of short duration—these are the most favorable cases, but I had better say these are the least undesirable cases to have come to us. The subsequent development of motion in the jaw is one thing that we have to take very good care of. Occasionally, after one divides the muscular attachments on both sides of the lower jaw, as is done where there is a fibrous ankylosis, a dropping of the jaw is the result, and that is quite annoying.

The next question is, What is the nerve-supply of all the muscles that are going to hold this jaw up subsequently? The jaw is moved by the masseter, by the pterygoids, and by the temporal. The internal and external pterygoids slide the lower jaw forward and sideways. The internal pterygoid is one of the powerful factors in holding the jaw up. It is assisted ably by the temporal and masseter muscles.

The nerve-supply comes from where? Not from the facial, but entirely and exclusively from the inferior maxillary. That means the inferior maxillary division of the fifth, which is a mixed nerve. It is largely sensory, but has a motor element, that is for the sole purpose of supplying these muscles of mastication, the temporal, masseter, and internal and external pterygoids. We will not come in contact with that nerve in doing our work at all. As it comes out of the skull through the foramen ovale, the muscular portion divides into two branches—an anterior and a posterior. Before division it supplies the internal pterygoid, and after division it is distributed to the other muscles of mastication. There is another nerve, one which has nothing to do with mastication, which may be involved in this particular field because it is exposed to being traumatized in work on the head, in this region. I refer to the facial nerve, which comes out of the skull through the stylomastoid foramen, and passes forward across the lower portion of the parotid gland, to be distributed to all the muscles of expression. There is danger of injuring that nerve and getting a permanent paralysis of these muscles of expression in the face, which would be a very serious thing—not so serious in this case as it is in the average case, because this boy has already a very badly disfigured face.

Then, again, there is another element of danger—that you cannot divide that bone at the original articulation, and you must not endeavor to try; if you do, you will go through the base of the skull. It is a very thin piece of bone. So we have planned in this case that if there is a bony ankylosis of the temporomaxillary joint the result of infection, we will cut off the condyle at the neck, clean out the glenoid fossa carefully, but only enough to make a fair cavity, and put in between these bony surfaces a flap of the aponeurosis of the temporal muscle, which is right in the field and therefore easily accessible. This is my plan. It is the best plan.

Now we will commence and do the operation. The incision will extend along in front of the ear, on the cheek, from just above the zygoma downward about two inches. We will keep to the hair-line fairly well, and anterior to the ear, so as to come down on the articulation at once. Then we will divide the zygoma and throw it forward out of our field. That exposes the joint at once. It is fixed. It is apparently only a fibrous ankylosis, the result of firm and continued muscle action. We will free all the temporal attachments from the bone without getting in the facial nerve zone at all, or we will remove the coronoid process, if we wish, and let the end of the cut muscle retract. We always have the pterygoids to fall back on to hold the jaw up.

In the skiagram of this case nothing definite can be outlined. There [indicating] should be the articulation, and if the trouble had been recent and on one side only, we would not have to reckon on the other side. Whether we shall have to operate on the other side depends on how much trouble we have with this side.

The Sister has to give the anesthetic in this case through the nose; there is danger of nose-bleed and the patient is unable to swallow.

VISITING PHYSICIAN: Doctor, what is your opinion of nitrous oxid gas as an anesthetic?

DR. MURPHY: We use it quite a little. Of course, ether is our favorite anesthetic here. Whenever we have an intestinal obstruction; whenever we have any desire to have the patient come out immediately from under the anesthesia; wherever we do a minor operation—we use nitrous oxid oxygen. We start every case with nitrous oxid oxygen. The minor operations we carry through with it, if we desire. But our anesthetic here is ether. It has been very satisfactory to us, and I think the reason is that we have had one party giving it for many years, and that that party is not a doctor, but a religeuse, which is a very great advantage in giving an anesthetic. The Sister sees the patient the night before the operation; she sees to the preparation of the patient, looks after the urine examination, sprays the throat, gives orders for and looks after the patient after the operation. Her opinion as to the patient's condition is more valuable to me than that of any one else.

VISITING PHYSICIAN: What about nerve block?

DR. MURPHY: We do not use it. In Europe this summer I saw all that variety of anesthesia and saw the various methods used splendidly. If you ask me now, after seeing all that, if I had to have an operation on my knee-joint would I take local nerve block or ether, I would say I would take ether. I could say this in justice, that with three exceptions ether was given badly. The Europeans do not give ether as well as we do here. I think that tells the story, and I think that is one of the reasons why we prefer ether.

We have no objection to any one using any of these methods, but we want it distinctly understood what we use, and why we prefer it. Ether, to my mind, is the best anesthetic for an operation. It is not the only anesthetic at all. Chloroform, I think, should not be given at all. I speak from experience. I had ten years of chloroform,—nothing but chloroform,—and never had a death, and in one case we kept up artificial respiration for three hours and fifteen minutes and saved the patient, but all one has to do is to read the daily papers and read records of chloroform deaths.

I have made my incision, as previously mentioned, cutting just in front of the hair-line, and keeping anterior to the temporal vessels, and well above the facial nerve. Next, I wish to provide for my interposing flap. I am going to take that flap from the temporal fascia. I am cutting very carefully because the temporomaxillary articulation is just beneath the zygoma. The zygoma has been severed and turned out of my field.

There is the main portion of that temporal muscle passing down beneath the zygoma to the inner side of the coronoid process. On further examination, with the joint freely exposed, I find that there is no bony ankylosis. That shows that this case is of the type I just told you about. The case I mentioned was not a bony ankylosis at all. It was a contraction of the fascia. Now, this is a much more difficult proposition to deal with—very much more difficult.

Now, then, as we have figured this out we may have to take off the head of the coronoid process because we may not be able to elongate the soft parts sufficiently to permit of motion in the joint. We will first detach the temporal muscle from the coronoid process, and this is what I am going to try to do, reaching through the sigmoid notch without disturbing the facial nerve. You have always to keep that facial nerve in mind. The temporal muscle we could loosen from its attachment to the skull and slip it down, if necessary. But we will

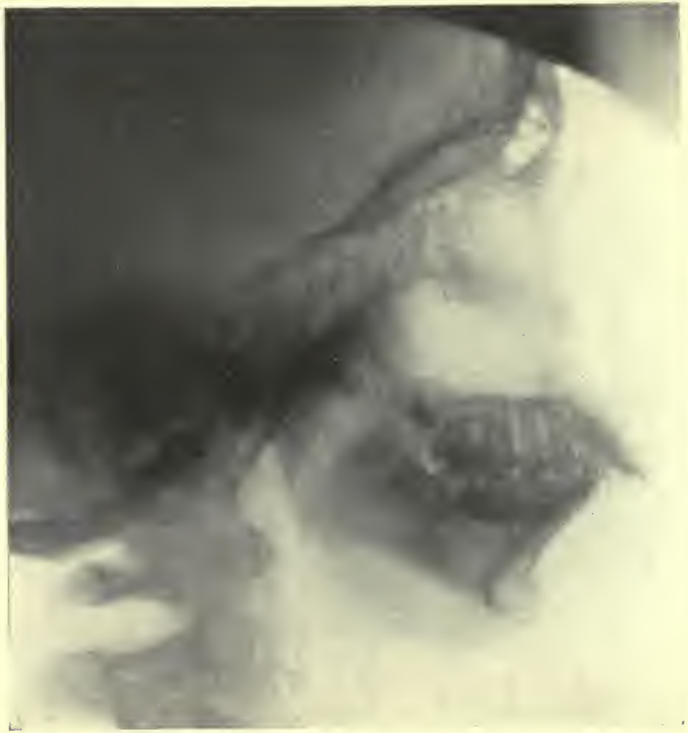

Fig. 163.—Skiagram of right side of head with complete bony ankylosis of the temporomaxillary joint.

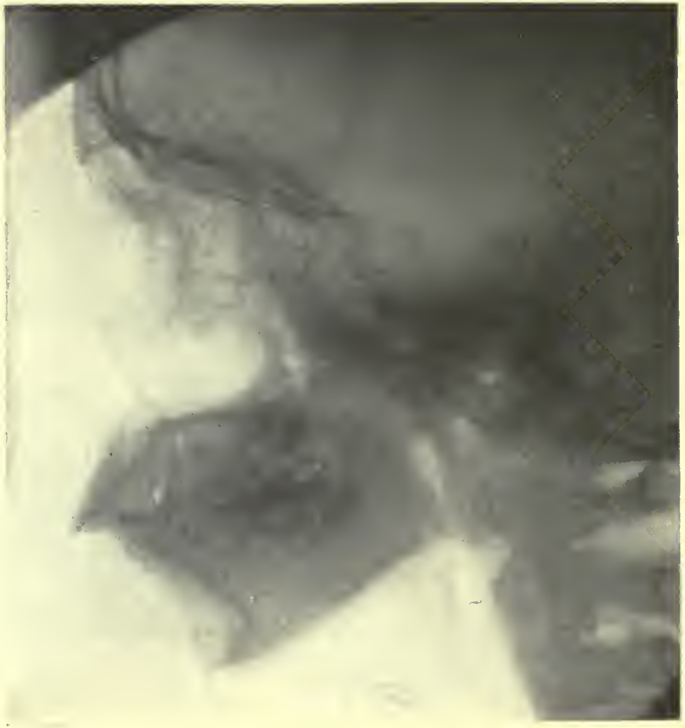

Fig. 164.—Skiagram of left side of head, to which all the symptoms in the case pointed as the seat of the trouble. At the operation no bony ankylosis was found, but a fibrous fixation.

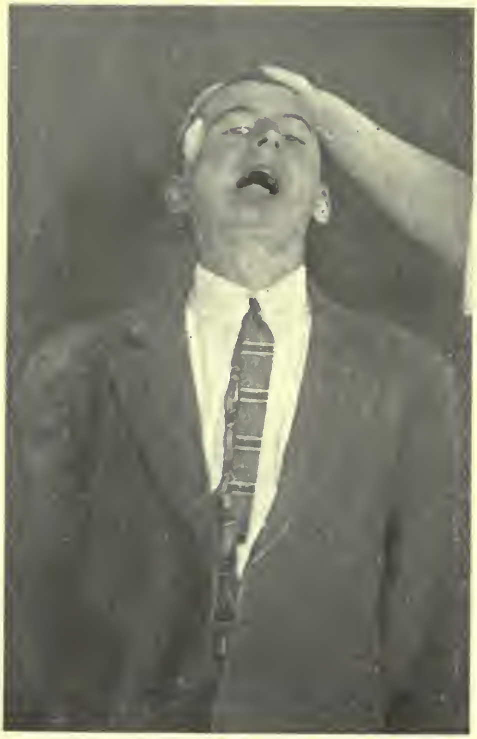

Fig. 165.—Photograph showing extent to which mouth could be opened after operation. Patient could insert two fingers and had free voluntary movement of the jaw.

loosen it from its attachment to the coronoid process, and then after you have the internal pterygoid loosened—only one—you do not have to figure on the other one at all, because the external pterygoid has no power at all except that of drawing the jaw forward and nothing to do with drawing the jaw upward.

There is where I believe he had his suppuration, in this glenoid cavity. There is the temporal muscle. What an enormous muscle that is! I divide that across at its attachment to the coronoid process, and then go down in the jaw and free the masseter and the internal pterygoid.

I am on the inner surface of the tip of the coronoid process and freeing that. It is freed. I bring my knife clear across now. The temporal muscle falls right back into position and needs no fixation.

This jaw is deformed. It has fallen down and has been pulled away back. You saw that the moment I exposed the joint it was perfectly free. I will now make an incision just below the angle of the jaw, so as not to have a scar on his face, and free the masseter from its attachment, as well as the internal pterygoid muscle just below the angle. Here is the attachment of the masseter standing right up on the bone. I am freeing that. We have the masseter off; now we must go in and get the internal pterygoid. The pterygoid goes right high up. Now I am getting on the internal pterygoid and am detaching it. It is perfectly free on the under side, and I have not disturbed the inferior maxillary nerve or vessels.

Now I have freed, as far as I can make out, the three important muscular attachments that hold that jaw up. Now I am going around into the mouth and see what is there. There is not a particle more motion here than there was before—not the slightest motion. I can put my fingers back the same as I could before, but there is no more motion. The jaw is just as firm and unyielding as it was before. That comes from the fascia and fascial contraction. I can feel the end of the coronoid process here. I think I have all that temporal muscle freed. If I had not been in this dilemma before I would be surprised.

Now there are two things left to do—one is to divide the coronoid process (I have not closed the incision above), and the other is to take off the condyle of the maxillary bone, but before doing that I will expose the other side.

There is not the slightest effort at motion on this side. This jaw ought to drop. I will expose the joint on the other side. Remember, he has no history of disturbance on the other side, but it has been fixed since he was seven months old. The question is, Is there a bony ankylosis on the other side? I believe there is. We can take out both condyles of the maxilla and let the jaw go up and down, and give him a means of opening his mouth. He must open his mouth. Nothing else will do.

We expose this joint in the same way as we did the other side. I remember one case in which I took off both the condyles and the jaw dropped clear down, and we had to block it on the chest for a long time to keep the patient from strangling to death when his tongue dropped down.

Here, you see, this jaw stands out prominently. It is not sunken in on this side. This jaw shows no gross trophic changes, so far as we are able to make out. This jaw should not have a bony ankylosis. From the history he never had a disease on this side. If he has an ankylosis, it is merely a static ankylosis, which was described in our article on Surgery of the Bone in 1896.

We are going down here onto the zygoma again. There is the temporal muscle; there is the margin of the zygoma, and I am keeping high up on account of my desire to protect the facial nerve. I am right on the hair-line with my skin incision. This is the side on which there is said to have been no infection. This side, of course, has been permanently out of commission with the other side.

Here, to all appearances, is a complete bony union! I want you to see that. It is a solid bony union. That is a union clear across to the tubercle here of the zygoma—one solid bony mass. The parents must have forgotten which side was affected when the boy was seven months old. Our findings show conclusively that it was the right side and not the left. We shall divide that with a chisel, keeping in the line of the old articulation. That clears it all up, and explains why the jaw would not come down when we freed the muscles on the other side. I must get that neck of the bone freed. It must be perfectly free. This ossification appears to come almost as far forward as the coronoid process.

Now I am anterior to the coronoid process. There is the coronoid process, and there is the bony union of the coronoid process to the base of the skull. That accounts for the firm fixation. It is clearly in view now. There is the posterior margin of the bone. We will take off the condyle of this bone. Now it is free. The mouth opens now. Before it would not open at all. There is free motion here now. It was not only an ankylosis of the condyle, but also of the coronary process.

Now, you see, we have got to insure against a reunion of this bone. He must be watched. There is a bare possibility of his swallowing his tongue when his jaw drops, though I do not think so. Now we have to make a non-union. I am preparing the interposing flap from the temporal fascia and fat. I must put the flap clear across that process of bone, otherwise he will get reëstablishment of the bony union. The base of the flap will be upward; the tip, down. This tip will be swung down into the joint. This is my regular procedure now.

This case is interesting in this fact: first, his ankylosis is on the side opposite to the one in which he was said to have had his infection, and there was not only ossification of the joint on that side, but ossification of the tip of the coronoid process. That brings out a histologic reason. Ossification occurs in certain white fibrous structures in the embryo, and these self-same structures have a tendency to ossify in postnatal life. This extended right straight along up until the zygoma and coronoid process had ossified. The flap is fastened in place with a few chromicized catgut sutures to the temporal muscle at its attachment to the coronoid process. That covers over all the raw bony surface and will prevent reunion. A few catgut stitches close the deeper portions of the wound, and the skin incision will be closed with horsehair. We will dust the wounds with bismuth subiodid.

A collodion gauze dressing is then used to seal the incisions, and an outer dressing is applied.

The greatest deformity occurred in the horizontal plane of the left half of the mandible. It was depressed and displaced inward. Just why that should occur is not the question. I am going to fill it out by transplanting a piece from his tibia on to the anterior external surface of his jaw, so as to bring it out to a normal anatomic line with the upper half of his face. This operation will not be done for two or three months.

[On the morning of the next day the patient was able to open his mouth far enough to introduce two fingers. His further progress was rapid and continuous. He had motion in his jaw,—free and voluntary,—and no pain at any time. The horsehair sutures were removed on the sixteenth day after the operation.—Ed.]

COMMENTS ON CASES PREVIOUSLY OPERATED ON

PERFORATIVE APPENDICITIS

I WILL ask the intern to read the history of a case of appendicitis on which we operated a day or two ago, in order to bring out one point in connection with it that is very important. [History read.] The leukocyte count was 28,000. The temperature was 101.6° F. Ten weeks ago she had an attack from which she recovered in three days. It did not look very much like an attack of appendicitis. We operated on her at 12 o'clock, twenty-four hours to the minute after the onset of pain. When we opened the abdomen there was a flow of pus. As I displaced the omentum and intestine to get at the appendix, a greater quantity of pus escaped. The appendix was free from organic adhesions, except at the tip, from the old attack. I lifted it up, put on vulsellum forceps to hold it, and there was no perforation of the appendix that I could recognize. I believed that the infection in the peritoneum which caused the pus was due to infection passing through the wall of the appendix.

After I removed the appendix I examined it, and on the mesenteric under side was a clean-cut perforation into the free peritoneal cavity. There were no adhesions around the opening, so that the pus was free in the cavity. When we took out the appendix and inverted the end, as we do in the intermediate operation, we put a drainage-tube, with many holes, into the pelvis, and the pus flowed out freely.

One thing we noticed before we discovered the perforation of the appendix was the fiery red surface of the small intestine. I would not say that the endothelial covering was rubbed off, but the bowel was very red and congested. The points which I wish to make are, first the high leukocyte count, indicating the degree of infection. Second, that the pus began to flow out as soon as we entered the peritoneal cavity, but it had no odor, which made one think that it was defensive pus, instead of destructive pus, which always comes as the result of a perforation. We put in a drainage-tube, as I have said, and closed the wound up to the drainage-tube, put the patient in a sitting position, and this morning the temperature was 100.4° F.; pulse, 104. She has had no emesis since the operation, and it looks as though she was going to get well.

We have in the hospital three cases of peritonitis of the perforative type. I did not class this case as belonging to the perforative type until I looked at the specimen.

INTUSSUSCEPTION

The other case I wish to call your attention to is one I saw yesterday afternoon at about 5 o'clock—the baby boy of one of my doctor friends. The child had been perfectly well up to 2 o'clock. At 2 o'clock it did not have the sudden severe crying spell that these patients usually have in cases of intussusception. You know, in the beginning of an intussusception there is a screaming from pain that is practically pathognomonic of the lesion. The patient will be sitting at the table, and suddenly double up with pain, get pale, and often vomit. So characteristic is this picture that you can readily make the diagnosis. In this case the onset was a little different. The child began to strain, later it vomited, and within a short time a tumor could be felt in the right side of the abdomen. Fortunately, Dr. Chancellor, a celebrated specialist in diseases of children, was living in the same house as the patient. He examined the child, and at once made a diagnosis of intussusception. The child was brought to the hospital, and we were able to make a reduction by pushing the intussusceptum out of the intussuscipiens from behind—milking it out, as it were. We never made traction on it, but milked it out from behind, which is our custom in these cases, and put in lateral sutures to prevent reinvagination. We did not have to make a dissection of the ileum because a positive diagnosis was made so early, for which Dr. Chancellor deserves great credit.

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